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REVIEW ARTICLE

Memory impairments in depression

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Abstract

Depression is characterized by persistent depressed mood, loss of interest or pleasure in previously enjoyable activities, recurrent thoughts of death, and physical and cognitive symptoms. Godlewska and Harmer's cognitive neuropsychological theory posits that cognitive dysfunction constitutes a core pathophysiological feature of depression, manifested through negative cognitive bias, memory impairment, and executive dysfunction. Prior studies showed that depression impairs memory, correlating with the severity of depressive symptoms. The hippocampus and cortex critically mediate memory encoding, retrieval, activation, and consolidation. Neurally, depression involves reduced hippocampal activation, prefrontal executive control dysfunction, hypoactivity in emotion-regulating regions, diminished cingulate gyrus activity and connectivity, and amygdala abnormalities. Memory impairment is linked not only to depression but also to systemic inflammation from physical diseases, which may disrupt central nervous system function and contribute to cognitive deficits. To elucidate memory impairment mechanisms in depression and guide precision therapies for cognitive rehabilitation, in this review, we introduce the psychological model and measurement tasks of memory and discuss different types of memory impairment in depression.

Keywords: Memory; Impairment; Depression; Somatic diseases; Hippocampus

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1. Introduction

Depression is a chronic and recurrent psychiatric condition characterized by depressed mood, social isolation, and anhedonia.[1] In addition, depression is a common and burdensome severe mental disorder, which is expected to become the leading cause of disease burden worldwide.[2,3] People with depression suffer from a decline in quality of life due to the disorder itself as well as related medical comorbidities, social factors, and impaired functional outcomes.[4] In 2021, Godlewska and Harmer[5] expanded and refined the cognitive neuropsychological model and proposed the cognitive neuropsychological (CNP) theory, which integrates physiological, social, and psychological factors, providing a theoretical framework for the understanding of the delayed onset of antidepressant drug effects. The point of focus for the CNP model is the negative bias in the processing of emotionally salient information in depression.[5] In addition, the CNP theory emphasizes the importance of interaction with the social environment.[6] In addition,

depression is primarily linked to three neurobiological mechanisms: reduced serotonin neurotransmission, the hypothalamic-pituitary-adrenal axis dysfunction, and impaired hippocampal dentate gyrus neurogenesis. Memory impairment is the most prevalent cognitive deficit in both depression patients and animal models.[7] As a pivotal component of the brain's executive control systems, memory function serves as the critical interface between information encoding, storage, and utilization. Through highly integrated hippocampal-prefrontal circuits, it mediates multi-stage information processing through three fundamental mechanisms: dynamic information routing, real-time cognitive control, and adaptive behavioral output.[8-10] Notably, memory circuits functionally interact with limbic emotion-processing networks, and their dysregulation constitutes a core mechanism underlying impaired emotional homeostasis in depression.[11,12] Depressive patients' failure to update negative memories due to hippocampal-prefrontal dysfunction exacerbates rumination, representing both a disease marker and pathogenic mechanism.[13,14] Extensive evidence indicates the hippocampus plays a central role in memory within the brain parenchyma.[10,15] The hippocampus, a two-layered cortical gray matter structure derived from the entorhinal fold, comprises dorsal and ventral subdivisions. Positioned caudal to the amygdala, its dorsal region mediates spatial memory while the ventral portion primarily processes affective information. The seminal case of Henry Molaison, who developed memory deficits after bilateral hippocampal resection, first conclusively linked the hippocampus to memory, prompting research into memory formation.[16]

Memory refers to the cognitive process in which the brain encodes, stores, and retrieves information about objective matters, including memorization, retention, recall, and recognition. According to the duration of memory, it can be classified into transient memory, short-term memory, working memory, and long-term memory. Instantaneous memory differs from long-term memory, which divides into explicit memory encompassing episodic, semantic, autobiographical memory, and implicit memory. Memory deficits in rodent depression models mirror human depression pathology: impaired spatial memory (reduced Morris water maze performance) and episodic memory (object recognition deficits) reflect hippocampal-dependent dysfunction, while working memory (T-maze) and fear extinction deficits align with prefrontal-amygdala circuit disruptions.[17] Clinical evidence indicates persistent memory impairment across both acute and remissive phases of depression, with cumulative deterioration following recurrent episodes. These cognitive deficits significantly compromise patients' quality of life and may lead to psychosocial disability. A meta-analysis of n-back task performance reveals that while individuals with depression show preserved basic attention, they exhibit specific deficits in higher-order working memory components (updating, maintenance, retrieval) under increasing cognitive load.[18] A neuroimaging narrative review further supports this finding, suggesting that cognitive biases in depression may stem from central executive dysfunction affecting working memory processing, which is linked to persistent negative thinking and rumination, key neural substrates include the left dorsal anterior cingulate cortex, left dorsolateral prefrontal cortex (dlPFC), and default mode network regions.[19] The memory system serves as a central hub in the pathophysiology of depression, rather than a mere epiphenomenon, where the exhaustion of compensatory capacity in memory-related neural circuits triggers acute depressive episodes. Specifically, memory systems drive depression pathogenesis through maladaptive plasticity in the hippocampal-prefrontal-amygdala circuitry, manifested by synaptic pruning abnormalities and reduced neurogenesis.[20] Therefore, this review aims to integrate multiple dimensions of memory impairment in depression, summarize the connections and interactions between depression and memory impairment, provide a basis for in-depth understanding of the pathophysiological mechanism of depression, and offer a new perspective for understanding the social functions and clinical significance caused by memory impairment in depression.

2. Measurement and classification of memory

2.1. Long-term memory

Long-term memory, characterized by its enduring retention and unlimited capacity, forms the foundation for accumulating personal experiences, developing cognitive abilities, and shaping overall psychological growth.[21] Long-term memory encompasses extensive knowledge, a broad scope, and prolonged duration.[22] As a core brain function, long-term memory underpins all cognitive processes with its unlimited capacity, enabling lifelong retention of early experiences. Its representations rely on structural storage spanning hierarchical levels, from conceptual schemas to individual item traces.[23] Neurophysiological studies indicate that the hippocampus and medial temporal lobe mediate long-term memory in healthy individuals, relying on interactions with cortical regions. Low-frequency cortical oscillations and high-frequency hippocampal ripples are essential for this process. An animal study revealed that during spatial and non-spatial memory tasks, hippocampus-cortical network oscillations coordinate to consolidate memories. Notably, such coordination persists even without active memory demands, varying by age, memory intensity, and type.[24] Research shows the

hippocampus is essential for long-term memory storage and retrieval as well as new associative memory formation.[25,26] One study demonstrated that hippocampal-mediated long-range gamma synchronization couples memory engram cells across cortical regions, facilitating memory storage and retrieval. Notably, the experimental mice exhibited prolonged learning duration and slower acquisition rates compared to controls, reflecting severe memory impairment.[27] Behavioral studies using a naturalistic What-Where-When task revealed that depressed patients exhibit situational memory deficits, primarily due to impaired object memory and temporal binding ability compared to controls. These findings confirm the presence of context-dependent memory dysfunction in depression, which co-occurs with heterogeneous long-term memory impairments.[28] Depression selectively disrupts episodic memory, a critical long-term memory system. A meta-analysis demonstrated small to moderate depression-related deficits in episodic memory; deficits were more pronounced in older age, in clinical depression, and in those receiving pharmacological treatment, while fewer deficits were observed in memory for negative material and with higher educational attainment.[29] Depression involves dysregulated neural circuits encompassing the hippocampus, amygdala, and frontal-limbic regions, which are critically engaged in memory processing. Episodic memory encoding, consolidation, and retrieval require phase-synchronized interactions between theta-gamma and sharp wave-ripple (SWR) across hippocampus-prefrontal cortex networks.[30,31] Depression disrupts hippocampus-prefrontal cortex oscillatory coordination, manifesting as attenuated theta phase synchrony, gamma power coherence, theta-gamma phase amplitude coupling, and SWR generation.[32] Dysregulation of monoaminergic and glutamatergic systems in depression directly contributes to domain-specific cognitive deficits. A Danish study found that depressed patients exhibited reduced 5-HT4 receptor binding in the inferior frontal, temporal, parietal, and occipital cortices compared to healthy controls. Untreated patients with moderate-to-severe episodes showed 7.0% lower binding, and verbal memory performance positively correlated with 5-HT4 receptor levels.[33]

An important cause of long-term memory impairment in patients with depression may be chronic stress-induced neuroinflammation. According to clinical evidence, various psychosocial stressors have been proven to accelerate the development of neuroinflammation and mental disorders. Persistent neuroinflammation, in turn, can induce depressive-like behaviors or promote the progression of depression.[10] In a subset of patients, chronic exposure to stress is an etiological risk factor for neuroinflammation and depression. Neuroinflammation affects up to 27% of patients with depression and is associated with a more severe, chronic, and treatment-resistant trajectory.[34] Most of the evidence demonstrating involvement of synaptic and cytoarchitectural/cytostructural changes associated with depression-like behavioral deficits comes from animal stress models, it is well known that chronic stress or chronic corticosterone (main stress hormone of the hypothalamic-pituitary-adrenal axis) administration can induce dendritic reorganization in the prefrontal cortex and hippocampus as well as reduction of the number of synapses.[35] Meta-analyses confirm that inflammation and neural abnormalities are particularly linked to adolescent depression onset in the context of early-life stress.[36,37] In the future, imaging techniques can be further adopted for the assessment of long-term memory in depression to clarify the relevant neural mechanisms, understand the cognitive mechanisms of depression, and develop targeted intervention measures.

2.2. Short-term memory, sensory memory, and working memory

Classical theoretical models suggest that visual short-term memory can be divided into two main memory systems: sensory memory, a short-lasting but high-capacity memory storage; and working memory, a long-lasting but low-capacity memory store.[38] Sensory memory, also known as instantaneous memory, refers to the brief retention of stimuli perceived by sensory organs, typically lasting about 1 s, in a landmark 1960 experiment, based on American psychologist Sperling's concept.[39] Short-term memory is also called short-term storage, primary memory, or active memory. The term indicates different systems of memory involved in retaining pieces of information, or memory chunks, for a relatively short time, typically up to 30 s.[21] Practically, short-term memory functions as a temporary scratchpad for recalling a limited amount of data, typically around 7 ± 2 items, in the verbal domain.[21] Depression involves dysfunction of the dlPFC, a recently discovered brain region that subserves working memory, abstraction, and the thoughtful regulation of attention, action, and emotion.[40] However, the dlPFC is very vulnerable to stress and inflammation, which are etiological and/or exacerbating factors for depression.[40] Neurobiochemical studies have found that depression is associated with abnormal synaptic transmission in the prefrontal cortex, and reduced expression of several synaptic genes has been observed in the prefrontal cortex of patients with depression. Autopsy of individuals who died by suicide after death revealed that the transcriptome sequencing technology (RNA-seq) of the dlPFC tissue also revealed a lower level of gamma-aminobutyric acid type A receptor.[41]

Short-term memory is crucial for higher cognitive functions, yet its storage capacity is severely limited. Thus, it

is necessary to selectively retain information relevant to our goals by controlling attention. This is facilitated by working memory, which consists of short-term storage and executive attention.[42] Working memory is a multicomponent system that is supported by overlapping specialized networks in the brain. Baddeley's working memory model includes four components: the phonological loop, the visuo-spatial sketchpad, the central executive, and the episodic buffer.[43] Working memory serves as a limited-capacity interface between sensory and long-term memory, integrating information from both to support cognitive tasks. Excessive negative emotional information consumes working memory capacity, exacerbating depressive symptoms. Research indicates that individuals with depression are more susceptible to interference from task-irrelevant information, particularly negative emotional stimuli. They struggle not only to prevent its intrusion but also to disengage from it in a timely manner. Consequently, excessive negative emotional information occupies working memory capacity, contributing to the onset and maintenance of depressive symptoms.[44] Depression is associated with deficits in working memory.[45] Several cognitive subprocesses interact to produce working memory, including attention, encoding, maintenance, and manipulation.[19] Studies employing the n-back task in depression reveals impaired processing speed and prolonged response times, though without significant group differences in accuracy, P2 amplitude, or theta event-related synchronization.[46] However, meta-analytic evidence demonstrates pronounced accuracy deficits under higher cognitive loads (peaking at 2-back), with age and clinical severity exacerbating these depression-associated cognitive impairments.[19] Collectively, depressed individuals dedicate greater levels of cortical processing and cognitive resources to achieve comparable working memory performance to controls. In the future, cognitive neuropsychology research could delineate the underlying neural circuits to inform targeted interventions for working memory deficits in depression.

2.3. Autobiographical memory

Autobiographical memory, a universal aspect of human long-term declarative memory, plays a pivotal role in psychological and interpersonal functioning. Growing evidence suggests its frequent involvement in rumination among individuals with depression. Autobiographical memory facilitates the enduring storage of personal life information by integrating both episodic ("I remember" experiences) and semantic ("I know" facts) components. The seamless functioning of autobiographical memory provides a sense of stability to both the self and the external environment, while enabling detailed future planning. Psychologically healthy individuals typically exhibit a positivity bias in autobiographical recall, which may contribute to mental well-being. Impairments in retrieving event-level, specific autobiographical memories, termed overgeneral memory, are recognized as a feature of clinical depression.[47] Poorer autobiographical memory may be a vulnerability for future episodes and improving autobiographical memory specificity could protect against relapse.[48] One well-established framework behind overgeneral memory is the "CaR-FA-X" model. This theory explains overgeneral memory in terms of three interconnected processes: capture and rumination (CaR), functional avoidance (FA), and reduced executive function (X).[49] An updated meta-analysis demonstrates overgeneral and specific autobiographical memory predict the course of depression.[47,50] Notably, a meta-analysis revealed that depression is associated with reduced memory specificity and increased categorical memory retrieval; these deficits were less pronounced in subthreshold and remitted cases, while overgeneral memory was consistently observed across all emotional valences.[51] Autobiographical memory involves the storage and retrieval of information from one's past and ranges from broad life periods down to the minute sensory details of a given event.[52] The successful recollection of events from one's past is critical to identity formation, problem-solving, and future goal direction.

2.4. Prospective memory

Prospective memory is a core neurocognitive ability that refers to memory for future intentions, such as remembering to take medications and to switch off appliances.[53] Any breakdown in prospective memory, therefore, has serious implications for the ability to function independently in everyday life. In many neurological disorders, prospective memory deficits are common even in the earliest stages and typically become more severe with disease progression.[54,55] Results regarding prospective memory function in patients with depression are inconsistent and require systematic investigation. A study utilizing portable functional near-infrared spectroscopy demonstrated that maintaining both social and non-social intentions engages broad activation in the medial and right prefrontal cortex (BA 10).[56] Notably, social intention maintenance specifically enhanced activation in the lateral prefrontal cortex (BA 45/46) compared to non-social conditions, highlighting the pivotal role of prefrontal regions in sustaining intentions and responding to prospective memory cues.[56] Event-based prospective memory refers to the ability to execute delayed intentions upon encountering specific cues; this capacity is impaired in depressed patients and impedes functional recovery, contributing to persistent cognitive deficits during remission.[57] This allows individuals with depression to experience varying degrees of cognitive

impairment during the recovery phase. Depression is associated with multiple neuropsychological deficits, including impairments in executive function, memory, and processing speed, which may hinder daily functioning.

2.5. Interaction between memory and emotion

Affective experiences are commonly represented by either transient emotional reactions to discrete events or longer-term, sustained mood states that are characterized by a more diffuse and global nature. While both have considerable influence in shaping memory, their interaction can produce mood-congruent memory, a psychological phenomenon where emotional memory is biased toward content affectively congruent with a past or present mood.[58] The study of interaction between memory and mood has direct implications for understanding how memory biases form in daily life, as well as debilitating negative memory schemas that contribute to mood disorders such as depression.[59] Hippocampus-dependent cognitive memory and dorsolateral striatum-mediated habit memory represent two distinct memory systems, both modulated by emotional arousal. Emotionally charged events are more likely to be retained than neutral ones.[60] Stressful situations significantly influence memory encoding, especially for emotionally charged stimuli. Theta oscillations, particularly those in the medial temporal lobe, play a pivotal role in this process. Similarly, studies on depressed patients have established a link between amygdala activity and emotional memory. When depressive individuals with impaired emotional memory received brain stimulation to enhance high-frequency amygdala activity, their symptoms showed significant improvement.[61] A magnetic source imaging study revealed that stress enhances memory-related theta oscillations, with particularly pronounced effects observed in the medial temporal and occipito-parietal regions. In addition, theta power increased in response to stress when memories were formed for emotionally negative stimuli instead of neutral ones.[62] This offers fresh perspectives on the brain processes that underlie the connection between stress, emotion, and memory.

3. Somatic disorders and memory

Memory is not only related to various mental illnesses, but it can also be impacted by physical illnesses. Obesity has now reached the status of a global health emergency. Growing evidence indicates that excess body weight correlates with multiple cognitive impairments, as well as structural and functional changes in the brain. Obesity-mediated inflammatory changes affect the physiological functions of the central nervous system, thereby possibly mediating the impact on various cognitive processes.[63] Previous studies have established a correlation between obesity and impairments in the frontal lobe and hippocampus, potentially leading to memory deficits. Empirical evidence further supports this association: a study found that individuals in the obese group had lower cognitive performance in tasks involving planning, decision-making, self-control, and regulation compared with participants of normal weight. This study provides empirical support for the relationship between obesity and cognitive decline, and highlights the potential impact of obesity on cognitive performance in women.[64] Evidence obtained from clinical and experimental studies shows that obesity may be associated with cognitive performance and executive function impairments; and inflammation, oxidative stress, insulin resistance, and hypertension act as mediators for the adverse effects of obesity on the brain.[65] Patients with diabetes mellitus exhibit an increased risk of developing dementia, though the underlying etiology is complex and partially attributable to genetic factors. In individuals with type 2 diabetes mellitus (T2DM), the hippocampus is identified as the most vulnerable brain region.[66] Notably, researchers have identified associations between polygenic risk scores and right hippocampal lymph node properties in T2DM patients, with these lymph node characteristics further correlating with episodic memory performance, demonstrating the existence of a gene-brain-cognition biological pathway.[67] It has been estimated that 20 – 70% of people with diabetes mellitus have cognitive deficits; high blood sugar affects key brain areas involved in learning, memory, and spatial navigation; and the structural complexity of the brain has made it prone to a variety of pathological disorders, including T2DM.[68] Cognitive impairment and subsequent dementia are considered significant health challenges, in patients with established dementia, it is argued that hypertension is the main risk factor for small vessel ischemic disease and additional cortical white matter lesions.[69] Cognitive domains and impairments associated with hypertension include learning, memory, attention, abstract reasoning, mental flexibility, psychomotor skills, and executive function.[70] Stroke survivors may not only face the challenge of physical disability but also the challenge of cognitive consequences. Post-stroke cognitive impairment has been associated with functional dependency and poorer quality of life. It has been found that 60 – 70% of stroke patients have perceptual dysfunction, executive dysfunction, abstract reasoning dysfunction, episodic memory, or language dysfunction.[71] The theory of hemispheric functional specialization refers to the theory that there are differences in the division of labor between the two hemispheres of the human brain (the left brain and the right brain) in cognitive, perceptual, and motor functions. It holds that the left and right hemispheres of the human brain have functional

differentiation and synergy in advanced cognitive functions. This theory predicts that the language working memory of stroke patients in the left hemisphere will be more severely impaired, while the spatial working memory of stroke patients in the right hemisphere will be more impaired.[72] A meta-analysis indicates that the working memory of survivors after stroke is generally impaired. All subsystems of working memory were significantly affected, and similar findings were also reported for non-verbal and verbal tasks. Extensive frontal and parietal network lesions lead to impaired working memory, which in turn results in a reduced ability to maintain both verbal and non-verbal information. Compared with the subacute phase, the effect size in the chronic phase is larger, and most longitudinal studies have shown that the working memory performance of stroke survivors has not improved.[71]

A large amount of clinical evidence shows that patients with concurrent physical diseases not only exhibit disorders in short-term memory encoding and retrieval but also have a significantly increased risk of long-term memory decline and dementia transformation. Future research needs to further clarify the causal pathways and explore the protective potential of intervention measures for underlying diseases (such as blood pressure control and anti-inflammatory treatment) in memory function, with the expectation of providing evidence-based evidence for interdisciplinary comprehensive management.

4. Conclusion

Depressed patients experience severe declines in quality of life due to impaired social and emotional functioning, physical pain, and persistent fatigue. These detrimental effects are further exacerbated by residual cognitive symptoms that endure beyond clinical treatment. This review synthesizes the multifaceted nature of memory impairment in depression from a neurocognitive psychology perspective, examines its potential consequences for social functioning, and discusses the contributions of comorbid physical illnesses to memory dysfunction. These insights deepen our understanding of cognitive health in depression and inform the development of targeted preventive and therapeutic strategies.

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Conflict of interest

The authors declare that they have no competing interests.

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