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Cognitive Neuroscience-Based Analysis of Memory Impairment Mechanisms and Therapeutic Interventions in Depression

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Abstract. Depression currently stands as one of the most prevalent mental disorders, with incidence rates escalating post-COVID-19 pandemic, constituting a pressing global public health priority. Memory impairment, recognized as a common cognitive deficit among individuals with depression, has emerged as a pivotal target for clinical diagnosis and intervention. This study focuses on elucidating the effects of structural and functional abnormalities within the prefrontal-limbic system circuitry of depressed patients on memory deficits, while concurrently evaluating the efficacy of various therapeutic interventions. The findings corroborate that depression induces neuroanatomical alterations in brain regions encompassing the hippocampus, prefrontal cortex, anterior cingulate cortex, and amygdala, collectively exerting negative feedback effects on depressive symptomatology. Although extant research partially corroborates the influence of monoaminergic systems on memory deficits, more robust empirical evidence is required to delineate their precise mechanistic pathways. Current therapeutic modalities exhibit moderate efficacy in addressing memory deficits, particularly autobiographical memory impairment. However, such intervention effects appear subject to significant individual variability and remain incomplete.

Keywords: Depression, Memory impairment, Cognitive neuroscience, Therapeutic interventions

1. Introduction

Depressive disorder, classified among the most prevalent mental disorders, is defined as a mood disorder characterized by persistent and significant low mood as the primary clinical feature, accompanied by varying degrees of cognitive and behavioural alterations. A subset of patients exhibits self-harm or suicidal behaviours, potentially leading to death . Memory impairment represents one of the most common cognitive deficits in depression, serving as a critical target for clinical diagnosis and intervention. Multiple meta-analyses have identified explicit memory bias as a hallmark feature of depression, manifesting as impairments across multiple memory domains including working memory, prospective memory, autobiographical memory, and episodic memory. For example, depressed individuals demonstrate preferential recall of negative memories over positive ones compared to healthy controls. The relationship between depression and memory

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impairment is mutually reinforcing. On one hand, depression induces measurable memory dysfunction; conversely, such cognitive deficits exacerbate depressive symptomatology. The predisposition toward negative memory retrieval perpetuates emotional distress, while impaired access to positive memories disrupts reward processing pathways, thereby reinforcing anhedonia – a core symptom of depression. Experimental evidence from animal models demonstrates that chronic reactivation of hippocampal cells encoding positive memories can effectively suppress depression-like behaviors [1]. This study therefore investigates memory impairment-related brain regions and therapeutic approaches in depression, with the aim of advancing clinical diagnostic and treatment paradigms.

2. Analysis of memory impairment mechanisms

The prefrontal-limbic system comprises the prefrontal cortex (PFC) and limbic structures (including the hippocampus, amygdala, and cingulate cortex), which form complex bidirectional circuits through multisynaptic connections, playing a critical role in memory formation and retrieval. For instance, the anterior cingulate cortex (ACC) and amygdala exhibit crucial functions in the encoding and extinction of emotional memory; the hippocampus and prefrontal cortex (particularly the dorsolateral prefrontal cortex, dlPFC) are pivotal for social memory and episodic memory consolidation; furthermore, the prefrontal-cingulate-hippocampal network significantly contributes to verbal and declarative memory processing. Therefore, structural damage within the prefrontal-limbic system exerts a deterministic role in mediating memory impairments observed in patients with depression.

Hippocampal structural and functional abnormalities in depression are closely linked to the mechanisms underlying memory impairment, as this brain structure plays a significant role in contextual, declarative, spatial, and episodic learning and memory. Numerous studies have demonstrated significant volume reductions in the hippocampus of depressed patients compared to healthy controls. A meta-analysis revealed an average 8% reduction in left hippocampal volume and a 10% reduction in the right hippocampus among individuals with depression [2], with right hippocampal atrophy showing significant correlations with the number of depressive episodes. Furthermore, research has identified weakened functional connectivity between the bilateral anterior hippocampus and the default mode network in depressed patients, whereas excessive connectivity between the posterior hippocampus and the posterior cingulate cortex may contribute to the persistence of traumatic memories [3]. These aberrant connectivity patterns are strongly associated with deficits in episodic memory retrieval observed in depressive disorders. Moreover, hippocampal neurogenesis is significantly impaired in individuals with depression. As demonstrated by Boldrini et al. [4], depressed patients exhibit reduced granule neuron density in the hippocampal dentate gyrus (DG) compared to healthy controls, with no significant difference observed between antidepressant-treated patients and healthy individuals. DG neurogenesis suppression has been closely linked to memory deficits [5]. This impairment in neurogenesis may be mechanistically associated with excessive glucocorticoid activation secondary to chronic stress-induced dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. At the epigenetic level, researchers have observed that chronic stress induces significant reductions in histone H3 acetylation levels at the promoter regions of BDNF, GDNF, and RAC1 genes within the hippocampus, thereby suppressing synaptic plasticity and neurogenesis. A study utilizing a chronic social defeat stress model identified reduced levels of H3K9ac, H3K14ac, H4K5ac, H4K8ac, H4K12ac, and H4K16ac in the dorsal raphe nucleus and hippocampus of mice [6]. These epigenetic alterations collectively impair memory function in individuals with depression.

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Beyond the hippocampus, other components of the prefrontal-limbic system also exhibit structural and functional abnormalities associated with memory impairment in depression. Multiple meta-analyses have identified volumetric reductions in limbic-associated regions such as the medial prefrontal cortex and ACC in depressed patients [7]. Postmortem studies further corroborate these findings, demonstrating reductions in neuronal size and cellular density within these regions [8]. Additionally, meta-analyses of functional magnetic resonance imaging (fMRI) data reveal aberrant activation patterns in the prefrontal and cingulate cortices during working memory tasks [9]. While these activation anomalies may reflect compensatory neural strategies for underlying dysfunction in depression, hyperactivation of the ACC has been hypothesized to interfere with goal-directed memory retrieval. This interference potentially contributes to clinical manifestations of ruminative thinking and fragmented memory recall. The amygdala is recognized as a central hub governing brain regions involved in memory encoding, consolidation, and emotional memory processing. Neuroimaging studies reveal structural abnormalities in depressed patients, with the right medial amygdala exhibiting significant volumetric enlargement compared to healthy controls. Young et al. [10] demonstrated through fMRI investigations during autobiographical memory recall that depressed individuals show reduced amygdala activation during positive memory retrieval and heightened activation during negative memory retrieval compared to non-depressed participants. These activation patterns strongly correlate with depressive symptom severity. This suggests a direct association between amygdala dysregulation and autobiographical memory impairment in depression. A recent fMRI meta-analysis focusing on autobiographical memory dysfunction in depression further implicates the amygdala and hippocampus as critical neural substrates of these deficits, potentially mediated through their interactions within the salience network and default mode network [11].

The serotonin and dopamine systems also play crucial roles in these processes. Accumulating evidence indicates that distinct serotonin receptors participate in learning and memory. Clinical studies in depressed patients have identified correlations between memory dysfunction and diminished 5-HT4 receptor binding in the brain [12]. Animal model studies demonstrate that chronic stress-induced reductions in serotonin concentrations impair hippocampal synaptic plasticity, thereby exacerbating memory deficits [13]. However, the precise mechanistic pathways through which serotonin dysregulation contributes to memory impairment in depression remain poorly characterized. In individuals with depression, memory impairment associated with positive events constitutes a hallmark feature of depressive symptomatology. The dopamine system, a critical component of the reward circuitry, plays a pivotal role in mediating such memory deficits. Midbrain dopamine neurons are established to encode quantitative reward prediction error signals, with recent evidence demonstrating their activation responsiveness to not only rewarding but also aversive stimuli. When higher-order cortical structures process signals from subcortical regions, synaptic connectivity remains maintained when signals align with prior expectations. However, exposure to novel stimuli that generate prediction errors induces synaptic plasticity modifications, thereby facilitating the encoding of new memories. Experimental studies utilizing animal models have revealed that localized inhibition of D1 dopamine receptors in the hippocampal CA1 region blocks late-phase long-term potentiation (LTP) while sparing early-phase LTP [14]. Chronic stress represents a prevalent etiological factor in depression. Notably, the dopaminergic system exhibits heterogeneous firing pattern abnormalities contingent on stressor characteristics: heightened tonic firing activity has been documented in specific stress paradigms [15], whereas attenuated phasic responses are observed in others [16]. However, empirical consensus regarding the mechanistic linkage between dopaminergic neuronal firing dynamics and depressive symptomatology remains

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elusive, necessitating further investigation to establish causal pathways. Furthermore, current research lacks empirical consensus regarding the directional relationship between dopamine neuronal firing dynamics and specific depressive symptom dimensions. Both serotonin and dopamine systems have been implicated in mediating cognitive deficits and memory impairments observed in depressive disorders. These neurotransmitter systems currently serve as primary pharmacological targets in clinical practice and demonstrate great clinical efficacy in symptom management. However, the precise mechanistic pathways through which these neuromodulatory systems interface with core depressive symptomatology remain incompletely characterized.

3. Analysis of intervention measures

3.1. Pharmacotherapy

Current therapeutic options for depression are highly diverse. As a core symptomatic feature of depressive disorders cognitive dysfunction constitutes a critical treatment target in antidepressant interventions. First-line pharmacological agents in clinical practice primarily include selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram, and escitalopram; serotonin-norepinephrine reuptake inhibitors (SNRIs), including venlafaxine, duloxetine, and milnacipran; noradrenergic and specific serotonergic antidepressants (NaSSAs), such as mirtazapine; and novel agents, such as agomelatine and vortioxetine.

A comparative efficacy study of four SSRIs (fluoxetine, sertraline, citalopram, and paroxetine) demonstrated that patients achieving symptom remission through pharmacotherapy exhibited superior performance on the Stroop test compared to controls [17]. However, conflicting evidence suggests potential memory impairments associated with fluoxetine treatment [18]. In a clinical trial involving adults with depression, citalopram administration over four weeks significantly improved Rey Auditory Verbal Learning Test (RAVLT) performance [19]. Concurrently, escitalopram was found to enhance emotional regulation, cognitive function, and facial recognition memory in elderly depressed patients during a randomized controlled trial [20]. Fluvoxamine is believed to improve cognitive memory by enhancing the division and proliferation of hippocampal cells [21]. Sertraline has also been found to have a positive effect on facial memory impairment [22], while another study observed that sertraline and bupropion had no effect on the recovery of emotional memory [23], and may even be detrimental to spatial memory [24]. Therefore, the efficacy of sertraline on memory impairment in patients with depression may need to be evaluated from multiple perspectives. In addition to the more commonly used SSRIs, duloxetine seems to have no effect on memory [25], while venlafaxine and agomelatine have shown preventive effects against memory impairment caused by chronic stress in animal models [26]. Collectively, SSRIs demonstrate a generalized ameliorative effect on memory impairment in patients with depression, albeit with varying efficacy levels and incomplete symptom resolution across individuals. A meta-analytic review supports these findings, indicating significant memory improvement linked to the use of SSRIs and SNRIs, whereas other classes of antidepressants have a more moderate effect [27].

3.2. Physical intervention

Beyond conventional antidepressant pharmacotherapy, physical interventions also exert significant effects on memory dysfunction in depressive disorders. Electroconvulsive therapy (ECT), a widely utilized physical treatment modality, has been reported to modulate emotional memory bias in depressed patients, yet paradoxically exacerbates memory dysfunction in certain cases. These

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discrepancies have been posited to be mediated by individual characteristics [28] and treatment intervals. For instance, Wiedemann et al. observed worsened autobiographical memory interview scores in patients undergoing memory reactivation one day prior to ECT sessions, whereas improved scores emerged when memory activation was separated from ECT administration by at least two days [29]. This temporal dependency suggests ECT may engage a disruptive-reconstructive mechanism on mnemonic processing, potentially informing optimized treatment scheduling in clinical practice.

Another common physical therapy intervention for depression is Transcranial Magnetic Stimulation (TMS). Compared to ECT, TMS exerts a less intense stimulatory effect on brain regions and has a weaker impact on memory function. A study demonstrated that TMS positively influences impairments in simple working memory tasks, while exerting no significant effect on more demanding memory tasks [30].

3.3. Cognitive therapy

In addition to pharmacological and physical interventions, psychotherapy constitutes a critical therapeutic approach for depression, particularly cognitive therapy within psychotherapeutic modalities. Cognitive therapy is believed to improve depressive moods and behaviors by focusing on the cognitive impairments of patients with depression. These impairments include impaired learning, memory function, and systematic negative cognitive biases. Emerging variants such as mindfulness-based cognitive therapy (MBCT) have been developed from cognitive therapy. A randomized controlled trial in late-life depression demonstrated MBCT's efficacy in alleviating autobiographical memory deficits [31]. However, the precise mechanisms underlying MBCT's effects on depressive relapse prevention and autobiographical memory modulation remain poorly characterized. A recent meta-analysis concluded that autobiographical memory specificity showed no significant improvement following MBCT interventions, with no evidence supporting memory specificity as a mediator of therapeutic outcomes [32]. These contradictory findings underscore the necessity for further empirical validation of cognitive therapy's purported benefits on memory impairment in depression.

4. Conclusion

This study systematically investigates the neurobiological mechanisms underlying memory impairment in depression and evaluates the intervention efficacy of current clinical treatments, thereby advancing comprehensive understanding of depressive pathology and informing diagnostic and therapeutic strategies.

The pivotal role of hippocampal dysfunction in depressive memory deficits has been extensively validated. Depression is associated with hippocampal volumetric reductions and suppressed neurogenesis, with pharmacological interventions demonstrating partial restoration of these pathological alterations. Crucially, such hippocampal impairments may reciprocally exacerbate depressive symptomatology, with emerging evidence suggesting mnemonic deficits might constitute a pathogenic mechanism in depression onset. Furthermore, meta-analytic evidence corroborates structural and functional abnormalities in prefrontal cortex, anterior cingulate cortex, and amygdala among depressed populations. At the molecular level, monoaminergic systems (serotonergic and dopaminergic) exhibit strong associations with memory dysfunction, as evidenced by the therapeutic targeting of serotonin pathways in SSRIs. However, the precise mechanistic contributions of serotonergic modulation to memory impairment remain inadequately characterized, while

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dopaminergic neuronal responsivity shows inconsistent correlations with clinical symptomatology. These empirical gaps necessitate subtype-specific consideration of monoaminergic influences, which may demonstrate differential regulatory patterns across depression subtypes and chronic stress paradigms.

Current first-line pharmacological agents demonstrate observable ameliorative effects on memory impairment in clinical populations, particularly regarding emotional and autobiographical memory domains. However, such therapeutic benefits exhibit substantial interindividual variability and often achieve incomplete symptom resolution. Among physical interventions, ECT shows relatively robust intervention efficacy, though its outcomes are potentially moderated by individual-specific factors and treatment interval parameters. The mechanistic underpinnings of ECT-induced memory modulation require further elucidation, which may inform future therapeutic protocols. In contrast, TMS displays limited efficacy in memory rehabilitation, with benefits largely confined to rudimentary memory enhancement. Cognitive-behavioral interventions targeting autobiographical memory deficits similarly necessitate further empirical validation of their therapeutic specificity.

This analysis primarily adopts a cognitive neuroscience framework. Future investigations should incorporate multidisciplinary frameworks to enrich theoretical and therapeutic perspectives.

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