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Common Neural Mechanisms Underlying Emotional and Cognitive Disorders: Circuits, Neurotransmitters, and Network Interactions

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Abstract:
Emotional disorders such as major depressive disorder (MDD) and bipolar disorder (BD), and cognitive disorders such as Alzheimer's disease (AD) and schizophrenia, have imposed an increasingly heavy burden on the global community and often occur simultaneously, which indicates that there are overlapping biological mechanisms. Current diagnostic frameworks remain categorical, and symptom based. This study examines shared neural mechanisms involving prefrontal–limbic circuits, large-scale networks, neurotransmitters, and neuro-immune–endocrine systems across these disorders. Evidence highlights convergent disruptions of the prefrontal–limbic circuit, including weakened top-down prefrontal control, hippocampal degeneration, and abnormal amygdala reactivity, which leads to executive, memory, and affective deficits. Whole-brain neuroimaging analyses implicate dysregulation of the default mode network (DMN), central executive network (CEN), and salience network (SN) as a unifying network signature underlying attentional dysfunction and impaired cognitive flexibility. At the neurochemical level, monoaminergic imbalance, dopaminergic dysregulation, and glutamate GABA imbalance collectively link mood instability with cognitive decline. Systemically, neuroinflammation and HPA axis hyperactivity disrupt neurotransmission, synaptic plasticity, and network function, which couples affective symptoms with cognitive impairment. These findings support a transdiagnostic model in which interacting circuit, network, and systemic abnormalities produce shared mechanisms across disorders. Recognising these common mechanisms may pave the way for improved therapeutics across emotional and cognitive disorders. For example, network-based biomarkers could enable earlier detection and precision diagnosis, while mechanism targeted interventions may reduce treatment resistance and enhance personalised outcomes.

Keywords: Transdiagnostic mechanisms; Neural circuits; Emotional and cognitive disorders.

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1. Introduction

Emotional and cognitive disorders are two major categories of psychiatric and neurological conditions, both contribute substantially to the global disease burden, which continues to increase.Emotional disorders, such as major depressive disorder (MDD) and bipolar disorder (BD), are among the most prevalent psychiatric illnesses worldwide. MDD is projected to become the leading cause of disease burden globally by 2030 [1]. It is characterised by persistent low mood, anhedonia, and impaired motivation, affecting over 300 million people worldwide, with the majority experiencing long-term disability [1]. Although BD is less common than MDD, it is highly disabling due to its recurrent episodes of mania and depression. Patients with BD usually experience fluctuating mood states, accompanied by long-term cognitive impairments [2]. Despite advances in pharmacological and psychotherapeutic interventions, up to one-third of patients with emotional disorders remain treatment-resistant [3]. Recognising these shared features is essential for advancing transdiagnostic approaches that move beyond diagnostic categories, particularly in relation to synaptic dysfunction and cognitive decline. Therefore, the aim of this study is to investigate the common neural mechanisms underlying emotional dysregulation and cognitive dysfunction across these disorders. This also aligns with recent frameworks such as RDoC, which emphasise cross-domain neural processes rather than symptom clusters.

Cognitive disorders, such as Alzheimer's disease (AD) and schizophrenia, which contributes to neurological disability. The most common symptom of AD is dementia, which affects about 55 million people worldwide and number of cases is expected to triple by 2050 [4]. It presents with progressive impairment of memory, deficits in executive function, and changes in behaviour. On the other hand, schizophrenia is a chronic psychiatric disorder characterised by diverse perceptual, emotional, and cognitive disturbances, including hallucinations and delusions [5]. Cognitive deficits in schizophrenia includes impairment in working memory, attention, and executive function, which strongly predict long-term disability and often persist after psychotic symptoms have improved.

Although mood and cognitive disorders are studied individually in detail, there is growing recognition of their overlap on clinical and neurobiological levels. Patients with AD have depressive symptoms are up to 40% of the cases. Those symptoms further exacerbate cognitive impairment [6]. Similarly, cognitive impairment is increasingly recognised as a core feature of emotional disorders. Deficits in attention, memory, and executive functioning persist during the euthymic state in BD and during remission in MDD [7]. In schizophrenia, comorbid depression is common and is associated with poorer functional outcomes. These patterns of comorbidity suggest that emotional dysregulation and cognitive impairment may arise from partially shared neural mechanisms rather than entirely distinct disease processes.

From a methodological perspective, converging evidence from neuroimaging and neurochemical studies supports this hypothesis. Both functional and structural neuroimaging have consistently identified abnormalities in prefrontal–limbic circuits across emotional and cognitive disorders [8]. Disruptions have also been observed in large-scale networks such as the default mode network (DMN), central executive network (CEN), and salience network (SN). Neurotransmitter dysregulation is typically described by the monoamine hypothesis of depression or the dopamine hypothesis of schizophrenia. However, recent studies have expanded the dopamine hypothesis to include glutamatergic and GABAergic imbalances, which are also identified as unifying features underlying impaired emotional regulation [9]. At the systems level, neuroimmune–endocrine interactions involving chronic inflammation and hypothalamic–pituitary–adrenal (HPA) axis dysfunction has been implicated in both depression and dementia, which suggests that peripheral immune activation can influence central neurotransmission and network activity [10].

Although numerous individual studies have examined the neural correlates of specific disorders, relatively few have systematically compared evidence across mood and cognitive disorders. Moreover, current diagnostic frameworks such as the DSM-5 remain categorical and symptom-based, often failing to capture overlapping biological mechanisms across conditions. Therefore, the aim of this study is to identify convergent neural circuits and neurotransmitter systems in both mood and cognitive disorders through neuroimaging, neurochemical, and systemic investigations. By highlighting shared mechanisms, this study seeks to contribute to the development of unified models of psychopathology that can guide future transdiagnostic interventions and personalised treatments.

2. Common Neural Circuit Mechanism

2.1 Prefrontal Limbic Circuits

The prefrontal–limbic circuit is a central hub responsible for regulating emotional as well as cognitive processes. The important components and structures include the anterior cingulate cortex (ACC), ventromedial prefrontal

cortex (vmPFC), dorsolateral prefrontal cortex (DLPFC), amygdala, and hippocampus. Evidence of dysconnectivity within this circuit across emotional and cognitive disorders has converged from resting-state fMRI and task-based paradigms. Overall, the prefrontal-limbic circuit is essential for emotional and cognitive regulation.

Patient with major depressive disorder (MDD), Jamieson found decreased connectivity from the amygdala to the DLPFC in response to negative emotional stimuli. Additionally, depressed individuals exhibited diminished functional connectivity from the amygdala to the rostral ACC during the processing of fearful facial expressions. This imbalance reflects weakened top-down regulation of emotional responding [8]. Tassone also conducted a functional neuroimaging study and revealed that increased amygdala activity during emotion processing was linked to increasing severity of MDD and negative bias. Diffusion tensor imaging (DTI) studies demonstrated a reduction in fractional anisotropy in white matter tracts connecting the PFC and amygdale, which further suggests structural underpinnings of these functional abnormalities [11].

Similarly, in the case of BD, Alahmadi found hyperconnectivity of the amygdala, during manic episodes, to be associated with increased emotional reactivity and emotional dysregulation. Increased connectivity is primarily found between the amygdala and vmPFC, but there is often decreased amygdala activity and connectivity during depressive states, which MDD shows a similar pattern [12]. This dysregulation of the amygdala-vmPFC circuit demonstrates how the same circuit can mediate different mood states in a similar disorder.

Schizophrenia involves dysfunction of the prefrontal–limbic circuit, evidenced by changes in the ACC, hippocampus, and amygdala. Kalin observed that the ACC was disconnected from both the prefrontal cortex and limbic regions, which caused an impairment on decision-making and resulted in negative symptoms. fMRI studies have reported that hyperactivity in the hippocampus results in elevated subcortical dopamine release via the striatum, which is associated with psychotic symptoms and executive function and contextual processing deficits. The amygdala was also described as being over-reactive to emotional stimuli, which showed the impairment of both the prefrontal cortex and ACC [13]. This dysregulation has implications for an increased perception of threat and inability to regulate emotional states. Furthermore, schizophrenia is further characterised by hypoactivation of the DLPFC during working memory tasks.

In Alzheimer's disease (AD), the prefrontal–limbic circuit is critically involved in executive function, memory, and emotion regulation. Sampath, Sathyanesan, and Newton reported that the hippocampus showed significant atrophy and metabolic decline, which leads to episodic memory formation impairments. Hippocampal degeneration also disrupts communication with the PFC, particularly the dorsolateral and ventromedial subdivisions, impairing higher-order cognitive control and decision-making. At the same time, the ACC shows reduced activity and connectivity, while the amygdala displays both volume loss and abnormal reactivity to emotional stimuli, which contributes to impaired emotional regulation. These abnormalities can be detected through volumetric MRI and functional connectivity analysis [14]. Collectively, such findings demonstrate that degeneration weakens the coordinated function of the prefrontal–limbic system.

In conclusion, mood and cognitive disorders share convergent dysfunction within the prefrontal–limbic circuit. Impaired prefrontal regulation disrupted ACC connectivity, hippocampal dysregulation, and abnormal amygdala reactivity emerge as common pathophysiological signatures. These disruptions impair the integration of cognitive and emotional processes, giving rise to overlapping features such as executive function deficits, memory decline, and emotional dysregulation.

2.2 Large-Scale Network Dysregulation

With the advancement of connectomics and graph-theory approaches applied to fMRI, research has shifted from studying individual circuits to examining whole-brain networks. Three large-scale networks have been consistently implicated across these disorders.

The default mode network (DMN) is involved in a variety of cognitive and affective processes, including emotional processing and experience recall. MDD is associated with hyperconnectivity within anterior DMN hubs, particularly the medial prefrontal cortex (mPFC). This increased internal connectivity is associated with a heightened reliance on self-focused maladaptive rumination. At the same time, the inability to regulate the connectivity between the DMN and task-positive networks results in decreased cognitive flexibility and attentional control, which also reinforces negative thought patterns [15]. Similarly, in AD, posterior cingulate cortex (PCC) and precuneus DMN nodes exhibit early metabolic dysfunction and impaired communication. The PCC and hippocampus are among the earliest sites of amyloid-β deposition and metabolic decline, which decreased connectivity across the posterior and medial temporal DMN nodes [15]. Thus, this highlights a role for DMN dysfunction in maladaptive self-focus in MDD and memory deficits in AD.

Central executive network (CEN) is a vital system responsible for executive functions such as attention, working

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memory, and decision-making. CEN is deeply established in dlPFC and posterior parietal cortex. CEN in BD is observed to have abnormal functional connectivities as well as reduced efficiency, specifically in executive regulation requirements. Neuroimaging evidence from Alahmadi shows hypoactivation of dlPFC while undertaking working memory and inhibitory control functions. These abnormalities are exacerbated with defective integration between CEN and DMN with failure in suppressing internal focus of attention when cognitive control is needed [12]. Moreover, fMRI in schizophrenia patients shows reduced connectivity in CEN, specifically between parietal hubs and DLPFC. Task-based fMRI also shows hypoactivation of DLPFC during cognitive tasks, which leads to deficits in goal-directed behaviour [16].

Salience network (SN), with origins in anterior insula and dorsal ACC, regulates switching between CEN and DMN activity. In BD and schizophrenia, the SN shows dysconnectivity between both networks. In BD, an emergent study of Alahmadi records that impaired SN connectivity disrupts its regulating function while switching between CEN and DMN, leading to inappropriate allocation of attentional assets and mood unstabilisation throughout the manic and depressive stages [12]. In schizophrenia, studies of Pietrzykowski similarly identify impaired SN connectivity, particularly decreased coupling with the CEN, which impairs efficient switching between internally and externally directed states. This dysfunction shows a deficit in working memory, attention, and screening out of irrelevant stimuli [16]. These findings point out the SN as a shared site of disorder between mood and cognitive disorder.

In summary, large-scale network dysregulation is a unifying pathophysiological feature for cognitive and mood disorders. DMN abnormalities cause maladapted self-referential processing in MDD and memory decline in AD, and CEN dysfunction is responsible for executive and attentional impairments in BD and schizophrenia. The SN functions as a dynamic switch between the DMN and CEN and is an overlapping site of disruption. This causes impaired allocation of cognitive and emotional resources across disorders. Overall, these findings indicate that disrupted interactions between DMN, CEN and SN drive overlapping deficits in emotion regulation, cognition, and behaviour, which provides a network-based framework for understanding transdiagnostic mechanisms and guiding the development of targeted interventions. These convergent disturbances imply that network-level biomarkers may be better transdiagnostic indicators than traditional symptom-based tests.

3. Neurotransmitter and System Interactions

3.1 Monoaminergic System

The monoaminergic system, including serotonin (5-HT), dopamine (DA), and noradrenaline (NA), is fundamental for mood regulation, motivation, arousal, and cognition. Dysregulation of these neurotransmitters is the primary focus of neurological study and is grounding for classic hypotheses, such as the serotonin deficiency hypothesis of MDD and dopamine hypothesis of schizophrenia. PET enables in vivo quantification of transporter binding and receptor availability, and MRS enables us to acquire knowledge about regional transmitter concentrations. In subsequent sections, we review convergent evidence for each system of neurotransmitters, with an emphasis on methodological approaches and cross-disorder relevance.

PET studies reviewed in Fakhoury show reduced serotonin transporter (SERT) binding in patients with MDD. Reduced binding is a marker of impaired reuptake activity, which leads to impaired homeostasis of the serotoneric system across cortico-limbic pathways, including the prefrontal cortex and hippocampus. These alterations affect mood regulation and negative affect [17]. Similarly, evidence reviewed in Saggu shows patients with AD have reduced serotonin and degeneration of serotonergic neurones in the cortex and hippocampus.These reductions impair synaptic plasticity and memory-related signalling, which contributes to progressive cognitive decline [18].

PET and single-photon emission computed tomography (SPECT) studies reviewed in Howes and Kapur indicate striatal presynaptic dopamine hyperactivity in patients with schizophrenia. Striatal hyperdopaminergia is correlative with the severity of positive symptoms. On the other hand, reduced dlPFC dopaminergic activity causes negative symptoms and cognitive impairments as well [19]. Moreover, Saggu report neuroimaging and post-mortem data indicating that dopamine is also responsible for depression, with reduced mesocorticolimbic activity causing anhedonia and defective reward processing. In AD, dopaminergic deficits in prefrontal cortex led to dysfunction of attention and apathy. Overall, these findings indicate dopamine dysfunction as a crucial mechanism correlating depressive motivational disturbances with cognitive decline in AD [18].

Furthermore, Saggu ehighlight that degeneration of the locus coeruleus in AD leads to widespread cortical and hippocampal NA depletion. This loss impairs attentional control, disrupts arousal regulation, and contributes to apathy and cognitive decline. There is also direct evidence

linking altered NA signalling with depression, where impaired stress responses contribute to cognitive fatigue [18]. In conclusion, convergent evidence demonstrates that monoaminergic dysfunction represents a transdiagnostic mechanism linking mood and cognitive disorders. Serotonin deficits contribute to mood dysregulation and memory decline, dopaminergic imbalance underlies both psychotic symptoms and motivational impairments, and NA depletion disrupts attention, arousal, and stress regulation in AD.

3.2 Glutamate-γ-aminobutyric acid (GABA) balance

Beyond with monoaminergic signaling, homeostasis between the excitatory neurotransmitter glutamate and the inhibitory neurotransmitter GABA is needed for the stabilisation of the neural networks and cognitive emotional regulation. Glutamate-GABA imbalance is a transdiagnostic process that is responsible for cognition, mood, and neurodegeneration. The following sections provide evidence throughout schizophrenia, MDD, and AD, and it reveals how convergent abnormalities in this system underlie shared symptoms.

In schizophrenia, as outlined in Chen et aexcitation and inhibition balance is disrupted in cortical circuits. Post-mortem and MRS studies show that reduced GABAergic signalling impairs gamma oscillatory activity that is critical for working memory and cognitive control. Abnormal vmPFC concentrations of glutamate are an additional disruption of cortical network functioning [20]. The imbalance produces cognitive deficits in schizophrenia.

In MDD, MRS studies by Sarawagi, Soni, and Patel reveal decreased GABA concentrations in prefrontal and occipital cortex, which impair inhibitory functioning. Conversely, hyperactivity of the glutamatergic system was associated with excitotoxic damage of hippocampal neurones. This is damaging to synaptic plasticity, network connectivity, and mood regulation [21].

In AD, evidence reviewed in Czapski and Strosznajder indicates that excessive glutamatergic activity, which includes NMDA receptor overactivation and impaired glutamate clearance. They promote excitotoxicity and synaptic loss, underlying memory impairment. Concurrently, deficits in GABAergic signalling, which includes reduced GABA concentration and downregulation of GABA receptors. These disruptions contribute to cortical hyperexcitability and disrupted gamma oscillations essential for cognitive function [22]. This imbalance therefore emerges as a central mechanism driving progressive cognitive decline in AD.

In conclusion, disruption of the glutamate-GABA balance represents a shared mechanism across mood and cognitive disorders. Despite disorder specific profiles, all three conditions converge on excitatory inhibitory imbalance as a central pathway to cognitive and affective symptoms.

3.3 Neuro-Immune-Endocrine interaction

Beyond neural circuits and neurotransmitter systems, interactions between the nervous, immune, and endocrine systems play a crucial role in the pathophysiology of emotional and cognitive disorders. Dysregulated immune signalling can alter neurotransmitter metabolism, impair synaptic plasticity, and disrupt network function, while endocrine factors such as cortisol mediate stress responses that directly impact limbic and prefrontal circuits. The following sections outline how inflammation and HPA axis dysregulation contribute to both mood and cognitive disorders, which highlight their shared role as transdiagnostic mechanisms.

Neuroinflammation emerges as a vital mechanism underlying cognitive decline in AD and mood dysregulation in MDD. In AD, activation of microglia and astrocytes in response to amyloid-β and tau pathology triggers the release of pro-inflammatory cytokines and complement factors. These processes impair synaptic function, reduce plasticity, and promote neuronal loss, which leads to progressive memory impairment and attentional deficits [23]. Similarly, in MDD, elevated levels of cytokines such as interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α) play a key role in pathophysiology. These cytokines also cross the blood–brain barrier, whereby they disrupt monoaminergic neurotransmission with reduced levels of serotonin and dopamine and intervene in glutamate–GABA imbalance with excitotoxicity. They also impair neuroplasticity and hippocampal neurogenesis, with consequences affecting mood regulation and cognition [24]. Thus, cytokine dysregulation is shared between MDD and AD.

Hypothalamic–pituitary–adrenal (HPA) axis is the body's primary stress-response system, regulating the secretion of cortisol for homeostasis and mood, cognitive, and arousal control. Belvederi Murri show BD is associated with dysregulated activity of the HPA axis and imbalanced secretion of cortisol. Patients have increased basal levels of cortisol and impaired negative feedback regulation, particularly in manic and depressive conditions [25]. Similarly, Sharan and Vellapandian indicate that HPA dysfunction is causal for cognitive deterioration in AD. Patients with AD exhibit elevated basal cortisol levels and impaired circadian regulation, resembling the abnormalities seen in BD. Prolonged HPA axis hyperactivity and cortisol exposure exert neurotoxic effects, particularly in the hippocampus, which leads to hippocampal atrophy, synaptic dysfunc-

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tion, and impaired memory formation [26]. These findings suggest that HPA axis dysregulation accelerates structural degeneration and cognitive decline in AD.

In conclusion, both neuroinflammation and HPA axis dysregulation act as transdiagnostic mechanisms linking mood and cognitive disorders. Elevated cytokine activity disrupts neurotransmission and synaptic plasticity, which contributes to mood instability in MDD and cognitive decline in AD. These findings underscore the shared role of immune–endocrine interactions in driving affective and cognitive pathology across disorders.

4. Conclusion

This study investigates the common neural mechanisms underlying emotional and cognitive disorders, which focus on shared dysfunction in circuits, neurotransmitter systems, and neuro–immune–endocrine interactions. Across these disorders, convergent abnormalities were identified in the prefrontal–limbic circuit, where impaired prefrontal regulation, hippocampal atrophy, and abnormal amygdala reactivity contribute to overlapping deficits in memory, executive function, and emotion regulation. Large-scale network dysfunction revealed by fMRI shows that disrupted interactions among the DMN, CEN, and SN represent a unifying framework of maladaptive rumination and impaired cognitive flexibility across MDD, BD, AD, and schizophrenia. At the neurochemical level, monoaminergic imbalance, glutamate–GABA imbalance, and dopaminergic dysregulation emerge as shared mechanisms linking mood instability with cognitive decline. Moreover, systemic processes such as neuroinflammation and HPA axis hyperactivity are evident in both emotional and cognitive pathology.

While prior studies have largely focused on isolated domains, this work highlights the critical role of transdiagnostic mechanisms in shaping our understanding of psychopathology. By acknowledging shared pathways, diagnoses can be refined and more precise treatments developed, with the potential to reduce high rates of treatment resistance, such as those seen in BD.

Future directions include the identification of network-based biomarkers through neuroimaging techniques such as fMRI, PET, and MRS. These biomarkers could help detect early disruptions in prefrontal–limbic circuits and large-scale networks, which offers more reliable transdiagnostic indicators and opportunities for early prevention. Additionally, neuroimmune and endocrine interventions, such as anti-inflammatory agents or HPA axis modulators, hold promise for simultaneously alleviating mood symptoms and cognitive decline. Therefore, identifying these shared mechanisms may facilitate the development of transdiagnostic therapeutic frameworks applicable across emotional and cognitive disorders.

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