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NEUROBIOLOGY OF DEPRESSION: ADVANCES IN NEURAL CIRCUITS, BIOMARKERS, AND IMPLICATIONS FOR MENTAL HEALTH CARE

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ABSTRACT

Depression is a complex, multifactorial disorder shaped by biological, psychological, and social determinants. This narrative review synthesizes contemporary evidence on the neurobiology of depression, emphasizing large-scale neural circuits and emerging biomarkers and discussing implications for diagnosis and care. Guided by a PICo strategy, we searched PubMed, Scopus, ScienceDirect, and SciELO for articles published between 2015 and 2025 in Portuguese, English, or Spanish. Eligible studies encompassed clinical, observational, experimental, translational, and review designs addressing neural circuitry, neurochemical and endocrine mechanisms, and candidate biomarkers. Findings converge on dysconnectivity across large-scale networks - default mode, salience, and central executive - alongside alterations within limbic–prefrontal circuits (prefrontal cortex, amygdala, hippocampus, cingulate, striatum) that underpin affective dysregulation, stress reactivity, rumination, and cognitive control deficits. At the systems level, chronic stress and hypothalamic–pituitary–adrenal axis dysregulation relate to hippocampal vulnerability, impaired neurogenesis, and symptom persistence. Biomarker research highlights pro-inflammatory cytokines (e.g., IL-6, TNF-α, CRP), reduced neurotrophic signaling (BDNF), and genetic/epigenetic profiles associated with severity and treatment resistance. Multimodal approaches integrating neuroimaging, perfusion metrics, and inflammatory panels show promise for risk stratification and prognosis, aligning with precision psychiatry. However, translation to practice is limited by clinical heterogeneity, methodological variability, and the absence of validated, clinically actionable biomarkers. Overall, integrating neural-circuit findings with biological markers and psychosocial assessment may refine diagnosis, personalize treatment selection (including neuromodulatory strategies), and shorten time-to-response. Future research should prioritize longitudinal, standardized, multimodal cohorts to validate biomarker panels and operationalize precision-guided care in routine mental health settings.

Keywords: Depression; Neurobiology; Neural Circuits; Biomarkers; Precision Psychiatry.

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1- INTRODUCTION

Depression is one of the most significant contemporary challenges in public health, characterized as a multifactorial condition that encompasses biological, psychological, and social dimensions. It is estimated that more than 300 million people are affected worldwide, representing the leading cause of global disability and exerting a substantial economic and social impact due to reduced productivity, functional impairments, and increased mortality, particularly related to suicide (GBD 2019 Mental Disorders Collaborators, 2022; Ilic; Ilic, 2022). Although historically understood from predominantly psychological or social perspectives, advances in neuroscience have broadened the understanding of the biological mechanisms involved in the development and maintenance of depressive disorders (Remes *et al*., 2021).

Over the past decades, research in functional neuroimaging, genetics, epigenetics, and neurochemistry has contributed to a detailed understanding of the neural circuits associated with emotional regulation, motivation, reward processing, and cognition - functions commonly affected in depression (Yankouskaya *et al*., 2022). Evidence indicates relevant alterations in structures such as the prefrontal cortex, amygdala, hippocampus, and striatum, as well as dysfunctions in the serotonergic, dopaminergic, and noradrenergic systems. In parallel, studies on the hypothalamic-pituitary-adrenal (HPA) axis, systemic inflammation, and neuroplasticity reveal a complex network of interactions that goes beyond the former neurotransmitter-centered perspective (Zhang *et al*., 2018; Filatova *et al*., 2021).

In this context, biomarkers have emerged as potential tools for early identification, prognosis, and therapeutic personalization in depression. Neurobiological, genetic, hormonal, and inflammatory markers have been investigated with the aim of improving diagnostic accuracy, predicting treatment response, and guiding more individualized interventions aligned with the paradigm of precision psychiatry (Elbakary *et al*., 2025). Such advances bridge clinical practice with basic science and foster a broader understanding of the disorder, contributing to the development of more effective and integrated therapeutic approaches.

In light of the above, this article aims to analyze the main advances related to the neurobiology of depression, focusing on neural circuits, emerging biomarkers, and their

implications for diagnosis and mental health care.

2- METHOD

This study is a Narrative Literature Review developed with the purpose of integrating and critically interpreting scientific evidence on advances related to the neurobiology of depression, with emphasis on neural circuits, emerging biomarkers, and their implications for diagnosis and mental health care. This type of review was selected because it allows for a broad, interpretative, and contextual analysis, articulating findings from neuroscience, clinical psychiatry, genetics, and related fields. The choice of this approach is justified by the multifactorial nature of depression and the heterogeneity of existing explanatory perspectives, which require a flexible, argumentative, and analytical format capable of fostering an integrated understanding of the different neurobiological phenomena involved (Rother, 2007; Green *et al*., 2006).

To guide the search and selection of studies, the PICo strategy was adopted, structured as follows: P (Population): Adults diagnosed with depression or under investigation for neurobiological mechanisms associated with depressive disorders; I (Interest): Scientific evidence related to neural circuits, biomarkers (neurochemical, genetic, inflammatory, and hormonal), and neurobiological mechanisms of depression; Co (Context): Clinical and laboratory settings of investigation in mental health, neuroscience, and psychiatry, including observational studies, clinical trials, and translational research.

Accordingly, the guiding question formulated for this review was: What current scientific evidence exists on the major advances in the neurobiology of depression, particularly regarding neural circuits, biomarkers, and their implications for diagnosis and mental health care?

The bibliographic search was conducted in the PubMed, Scopus, ScienceDirect, and SciELO databases, including publications in Portuguese, English, and Spanish, from 2015 to 2025. Controlled descriptors (MeSH/DeCS) and free terms related to the core axes of the review were used, such as: “depression”, “neurobiology of depression”, “neural circuits”, “biomarkers”, “inflammation”, “HPA axis”, “neuroplasticity”, “brain

imaging", "psychiatric biomarkers".

Included studies comprised clinical trials, systematic and narrative reviews, observational and experimental studies, and translational research addressing neurobiological aspects of depression. Opinion articles, duplicate reviews, studies exclusively involving pediatric populations, investigations focused solely on pharmacological treatment without discussion of neurobiological mechanisms, and studies that did not directly address evidence on biomarkers or neural circuits in depression were excluded.

After critical reading and comparative analysis of the content, the selected studies were organized into interpretative thematic axes, aiming to identify convergences, controversies, and gaps in the contemporary literature on the neurobiology of depression, thus enabling an integrative synthesis of recent advances and their contributions to clinical understanding of the disorder.

3- RESULTS

The analyzed studies demonstrate significant advances in the understanding of the neurobiological mechanisms involved in depression, revealing structural and functional alterations in brain circuits responsible for emotional regulation, motivation, cognition, and behavior. Research indicates dysfunctions in regions such as the prefrontal cortex, amygdala, hippocampus, cingulate gyrus, and striatum, with repercussions on neurotransmitter systems, particularly the serotonergic, dopaminergic, and noradrenergic pathways (Fang *et al*., 2025). In addition, functional neuroimaging studies have identified abnormal connectivity patterns among neural networks such as the default mode network (DMN), salience network (SN), and central executive network (CEN), supporting the hypothesis that depression reflects a functional imbalance between brain systems involved in self-referential processing, stress response, and emotional regulation (Du *et al*., 2025; Jiao *et al*., 2025).

The literature also highlights advances in the study of biomarkers associated with depression, with emphasis on inflammatory markers (IL-6, TNF-α, and CRP), alterations in the hypothalamic–pituitary–adrenal axis, neurotrophic factors (particularly BDNF), and genetic and epigenetic profiles related to vulnerability to depressive disorders.

Recent evidence suggests that reduced BDNF levels and epigenetic modifications in genes associated with stress response have been linked to chronicity and treatment resistance (Filatova *et al*., 2021; Du *et al*., 2025; Jiao *et al*., 2025). In parallel, contemporary findings reveal significant challenges, including the clinical heterogeneity of depression, the absence of validated biomarkers for routine clinical use, and the need for methodological standardization in studies that investigate correlations between neurobiology and clinical phenotypes (Zhang *et al*., 2018; Filatova *et al*., 2021).

To systematize the main findings of this section, a synthesis table was developed to summarize the most relevant evidence regarding neural circuits, emerging biomarkers, contributions to diagnosis, and gaps identified in the current literature on the neurobiology of depression.

Table 1: Key neural circuits, biomarkers, and challenges in the neurobiology of depression

Thematic Axis / Modality	Main Findings	Key Evidence (Authors/Year)
Large-scale network dysconnectivity (DMN, SN, CEN)	Aberrant within- and between-network connectivity linked to rumination, self-referential bias, and cognitive control deficits; patterns help distinguish subtypes and treatment resistance.	Barreiros et al. (2024); Fang et al. (2025); Rai et al. (2021); Yankouskaya et al. (2022)
Limbic–prefrontal circuitry	Functional and structural alterations in prefrontal cortex, amygdala, hippocampus, cingulate, and striatum associated with affective dysregulation, stress reactivity, and decision-making difficulties.	Fang et al. (2025); Du et al. (2025); Rai et al. (2021)
HPA axis dysregulation & stress biology	Chronic stress and HPA hyperactivation relate to hippocampal changes, impaired neurogenesis, and symptom persistence.	Lei et al. (2025); Kong et al. (2025)
Neurotrophic signaling & plasticity (BDNF)	Reduced BDNF and impaired synaptic plasticity associated with chronicity and poorer treatment response; plasticity-enhancing strategies are promising.	Yang et al. (2020); Toader et al. (2025)
Immune–inflammatory biomarkers	Elevated pro-inflammatory cytokines (e.g., IL-6, TNF-α, CRP) and immune–brain crosstalk correlate with severity and treatment resistance; cytokine shifts observed with somatic therapies.	Jiao et al. (2025); Elbakary et al. (2025); Gay et al. (2021)
Multimodal and translational biomarkers (imaging/genetics/perfusion)	Combining MRI/perfusion with inflammatory profiles improves risk stratification and prognosis;	Batail et al. (2025); Zhang et al. (2018); Barreiros et al. (2024)

	neuroimaging-genomics highlights biological heterogeneity.	
Clinical implications & neuromodulation	Biological profiling may inform personalized treatment selection; accelerated TMS and other neuromodulatory approaches align with plasticity models.	Van Rooij et al. (2024); Filatova et al. (2021); Cui et al. (2024)
Challenges and limitations	Clinical heterogeneity; lack of validated, clinically actionable biomarkers; methodological variability and standardization gaps.	Filatova et al. (2021); Remes et al. (2021)
Future perspectives	Integration of longitudinal cohorts, multimodal panels (inflammation–neuroimaging–genetics), and precision psychiatry frameworks to shorten time-to-response.	Batail et al. (2025); Cui et al. (2024); Barreiros et al. (2024)

Prepared by the author (2025) based on the reviewed literature.

4- DISCUSSION

Recent advances in the field of neuroscience have significantly broadened the understanding of depression beyond reductionist models focused solely on neurotransmitters, highlighting its configuration as a complex disorder involving neurobiological alterations, genetic vulnerabilities, environmental factors, and psychosocial processes. The findings of this review reinforce that the integration between basic science and clinical practice has fostered more comprehensive approaches capable of explaining the heterogeneity of the disorder and guiding more individualized therapeutic strategies. In this context, the discussion is organized into two analytical categories: (1) Neural circuits and pathophysiological mechanisms of depression, and (2) Emerging biomarkers and perspectives for precision psychiatry.

4.1 Neural Circuits and Pathophysiological Mechanisms of Depression

The analyzed literature shows that depression is associated with dysfunctions in neural circuits responsible for emotional regulation, reward processing, stress responses, and cognitive functions. Alterations in the prefrontal cortex, amygdala, hippocampus, cingulate gyrus, and striatum demonstrate the interconnection between emotional dysregulation and cognitive impairment, frequently observed in clinical practice (Barreiros *et al*., 2024; Fang *et al*., 2025). Furthermore, hyperconnectivity of the default mode network (DMN) and hypoactivity of the central executive network (CEN)

reinforce the presence of rumination, impaired decision-making, and a negative self-referential cognitive pattern - characteristic features of depressive symptomatology. These findings contribute to understanding depression not merely as an affective phenomenon, but as a disorder of brain networks (Rai *et al*., 2021).

In addition to structural and functional findings, the pathophysiological mechanisms of depression involve neurochemical and neuroendocrine alterations that modulate central nervous system functioning. Evidence highlights the role of the serotonergic, dopaminergic, and noradrenergic systems - associated with the regulation of mood, motivation, and stress response - which, when dysfunctional, contribute to anhedonia, hopelessness, and cognitive alterations (Cui *et al*., 2024; Lei *et al*., 2025). Chronic activation of the hypothalamic–pituitary–adrenal (HPA) axis under prolonged stress also plays a central role in the disorder's neurobiology, promoting hormonal imbalances, reduced neurogenesis, and changes in synaptic plasticity, particularly in the hippocampus (Kong *et al*., 2025).

Another relevant aspect highlighted by the studies concerns brain plasticity as an essential element in understanding the clinical course of depression. Dysfunctions in neuroplasticity mechanisms contribute to symptom maintenance, poorer therapeutic response, and increased risk of chronicity (Yang *et al*., 2020). The impairment of new synaptic connections, associated with reduced levels of neurotrophic factors such as BDNF, underscores the need for therapeutic strategies that stimulate the repair and reorganization of neural networks. In this regard, pharmacological, psychotherapeutic, and neuromodulatory interventions that promote plasticity have emerged as promising approaches (Van Rooij, 2024; Toader *et al*., 2025).

4.2 Emerging Biomarkers and Perspectives for Precision Psychiatry

Research on biomarkers for depression has intensified over the past decade, with the aim of improving diagnostic accuracy, predicting therapeutic response, and guiding more individualized treatments. The findings of this review show that inflammatory, hormonal, genetic, and neurochemical markers constitute an emerging and still developing field, reinforcing the potential of precision psychiatry (Elbakary *et al*., 2025; Filatova *et al*., 2021). Increases in pro-inflammatory cytokines and hormonal alterations

related to the HPA axis have been associated with symptom severity and treatment resistance, signaling the need to integrate biological assessment into clinical reasoning (Jiao *et al*., 2025; Gay *et al*., 2021).

From a practical perspective, the incorporation of biomarkers may help reduce the empirical nature of therapeutic decision-making, fostering a more personalized approach to care. Studies suggest that distinct biological profiles may be associated with clinical subtypes of depression, such as the inflammatory subtype, the subtype related to chronic stress, and that linked to alterations in reward circuits (Du *et al*., 2025; Filatova *et al*., 2021). Identifying these profiles, combined with tools such as neuroimaging and genetics, may assist in selecting more specific interventions, avoiding ineffective treatments, and shortening the time to therapeutic response - one of the greatest current clinical challenges (Batail *et al*., 2025).

However, despite advances, important challenges remain for the validation and implementation of biomarkers in clinical practice. Methodological heterogeneity among studies, lack of standardization in collection and analysis protocols, and the high cost of some examinations still limit their clinical applicability (Filatova *et al*., 2021). Furthermore, biological markers should not be interpreted in isolation, as depression is a multifactorial phenomenon that involves subjective and contextual dimensions (Remes *et al*., 2021). Thus, integrating neurobiological findings with psychosocial assessment remains essential for an ethical, humanized approach consistent with the principles of comprehensive mental health care.

5- FINAL CONSIDERATIONS

The present study demonstrated that depression is a complex and multifactorial disorder, deeply influenced by neurobiological alterations involving neural circuits, neuroendocrine mechanisms, and inflammatory and genetic processes. The analysis of the literature revealed significant advances in the understanding of the pathophysiological mechanisms underlying the disorder, as well as in the development of biomarkers with the potential to enhance diagnostic accuracy, predict therapeutic responses, and support more targeted interventions. These findings reinforce the relevance of integrating neuroscience into clinical practice, broadening the perspective

on depression and its multiple manifestations.

By highlighting the role of neural circuits and emerging biomarkers, this review contributes to the contemporary debate on the need for more personalized approaches to mental health care. The identification of distinct biological profiles and the understanding of functional and structural alterations in the brain networks involved in the disorder may guide more individualized therapeutic strategies, strengthening the movement toward precision psychiatry. Furthermore, the articulation between basic science and clinical practice represents an essential step for improving assessment and intervention practices, benefiting clinical decision-making.

Despite these advances, challenges remain that require further scientific and clinical investigation, particularly with regard to the validation and applicability of biomarkers in routine care. Future studies should prioritize standardized methodologies, representative samples, and integrative analyses that consider the biological, psychological, and sociocultural aspects of depression. The importance of care models that combine neuroscientific evidence with psychosocial approaches is emphasized, ensuring ethical, humanized, and person-centered practices. In this sense, a broadened understanding of the neurobiology of depression may contribute to the development of more effective strategies for prevention, diagnosis, and treatment, with positive impacts on the quality of life of affected individuals.

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