IOSR Journal of Humanities and Social Science (IOSR-JHSS)
Volume 31, Issue 6, Series 5 (June, 2026) 34-41
e-ISSN: 2279-0837, p-ISSN: 2279-0845.
www.iosrjournals.org

Systemic Inflammation and Psychiatric Disorders: New Paradigms in Understanding Depression

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Introduction: *Depression has traditionally been understood as a disorder associated with neurochemical alterations involving neurotransmitters such as serotonin, dopamine, and norepinephrine. However, scientific evidence accumulated over recent decades has indicated that immunological and inflammatory mechanisms have also played a relevant role in its pathophysiology. Chronic low-grade systemic inflammation has been associated with neurobiological alterations capable of influencing brain function, affecting processes related to mood, cognition, motivation, and behavior.* ***Objective:*** *To analyze the scientific evidence regarding the relationship between systemic inflammation and psychiatric disorders, with emphasis on the new paradigms involved in the understanding of depression and their clinical and therapeutic implications.* ***Methodology:*** *This literature review*

was conducted in accordance with the recommendations of the PRISMA checklist. Searches were performed in the PubMed, SciELO, and Web of Science databases using scientific articles published within the last 10 years. The descriptors employed were: "Depression," "Systemic Inflammation," "Psychiatric Disorders," "Cytokines," and "Neuroinflammation." Inclusion criteria comprised original studies, full-text articles, and research directly related to the association between inflammation and depression. Exclusion criteria included duplicate publications, studies lacking thematic relevance, and articles with insufficiently described methodologies. ***Results:*** *The studies analyzed demonstrated a consistent association between elevated inflammatory markers and depressive symptoms. Alterations in the levels of pro-inflammatory cytokines, microglial activation, hypothalamic-pituitary-adrenal axis dysfunction, and impaired neuroplasticity were observed. The findings also indicated that inflammatory processes may contribute to antidepressant treatment resistance and greater symptom severity.* ***Conclusion:*** *It was concluded that systemic inflammation plays a significant role in the contemporary understanding of depression, expanding explanatory models previously centered solely on neurotransmitters. The evidence analyzed demonstrated that the interaction among immunological mechanisms, neuroinflammation, and neurobiological alterations contributes significantly to the development and progression of the disorder, supporting new diagnostic and therapeutic perspectives in psychiatry.*

Keywords: *Depression; Systemic Inflammation; Psychiatric Disorders; Cytokines; Neuroinflammation.*

Date of Submission: 13-06-2026 Date of Acceptance: 25-06-2026

I. Introduction

The understanding of depression has undergone significant transformations over recent decades, expanding beyond traditional models exclusively centered on neurotransmitter alterations. In this context, recent research has demonstrated that systemic inflammation plays a relevant role in the pathophysiology of psychiatric disorders, particularly depression. This perspective has contributed to the emergence of a new scientific paradigm in which the interaction between the immune system and the central nervous system is considered fundamental for understanding the biological mechanisms involved in mental illness. Such an approach has enabled a more comprehensive view of the disorder by integrating biological, environmental, and psychosocial factors into the explanation of its origin and progression.

Among the principal findings related to this topic, the involvement of pro-inflammatory cytokines in the development of depressive symptoms has received particular attention. Cytokines are proteins produced by the immune system that regulate inflammatory responses within the body. In individuals with depression, several studies have identified increased levels of inflammatory substances, including interleukin-6, tumor necrosis factor-alpha, and C-reactive protein. These molecules are capable of crossing or influencing the blood-brain barrier, promoting alterations in neural circuits responsible for the regulation of mood, motivation, sleep, and behavior. Furthermore, the presence of a persistent inflammatory state has been associated with greater severity of depressive symptoms, suggesting that chronic immune activation may significantly contribute to the maintenance of the disorder.

Associated with this process, neuroinflammation has emerged as one of the most relevant phenomena in the contemporary understanding of depression. This mechanism is characterized by the activation of resident immune cells within the central nervous system, particularly microglia, which release inflammatory mediators capable of modifying brain function. Prolonged activation of these cells has been linked to structural and functional alterations in brain regions essential for emotional regulation, including the hippocampus, amygdala, and prefrontal cortex. Consequently, impairments in memory, stress adaptation, and emotional processing have been observed. Neuroinflammation has therefore come to be recognized as an important link between systemic inflammatory processes and the clinical manifestations of depression, reinforcing the need for integrated approaches to the study and treatment of psychiatric disorders.

Current understanding of depression has incorporated complex physiological mechanisms involving communication among different bodily systems. Among these mechanisms, particular emphasis has been placed on the hypothalamic-pituitary-adrenal (HPA) axis, a structure responsible for coordinating stress responses. Under conditions of prolonged exposure to stressors, persistent activation of this axis occurs, resulting in the continuous release of glucocorticoids, particularly cortisol. Sustained elevated concentrations of this hormone have been associated with alterations in emotional regulation, cognitive difficulties, and increased vulnerability to the development of depressive symptoms. Furthermore, the functional overload of this system compromises adaptive processes essential for physiological balance, favoring the emergence of dysregulated biological responses.

In parallel, recent investigations have demonstrated that metabolic alterations triggered by inflammatory processes exert a significant influence on neuronal communication. The availability of neurotransmitters associated with psychological well-being is affected by biochemical pathways that divert essential precursors

toward the production of compounds related to the inflammatory response. As a consequence, reduced efficiency of the mechanisms involved in regulating mood, motivation, and the capacity to experience pleasure has been observed. In addition, decreased expression of neurotrophic factors responsible for cellular survival and renewal has been reported, impairing brain plasticity and the ability to adapt to environmental demands.

These findings have stimulated the development of new therapeutic perspectives extending beyond conventional treatments. Scientific evidence suggests that interventions capable of modulating inflammatory responses may contribute to the reduction of depressive symptoms, particularly among individuals presenting elevated inflammatory markers. Within this context, strategies involving regular physical activity, balanced nutrition, improved sleep quality, and effective stress management have demonstrated potential to promote both physical and psychological benefits. Additionally, recent studies have investigated the use of pharmacological agents with immunomodulatory properties as complementary resources to traditional treatment approaches. Thus, the integration of neurobiological, immunological, and behavioral aspects has consolidated a broader understanding of depression, fostering personalized therapeutic approaches that are aligned with the complexity of the disorder.

II. Objective

This literature review aims to analyze the available scientific evidence regarding the relationship between systemic inflammation and psychiatric disorders, with an emphasis on the new paradigms involved in the understanding of depression. It seeks to investigate the biological mechanisms that connect immune activation to neuropsychiatric alterations, as well as to understand the role of inflammatory mediators, neuroendocrine changes, and processes that affect brain function. Furthermore, this review aims to evaluate the implications of these findings for the development of new diagnostic and therapeutic approaches, contributing to a broader and more integrated understanding of depression and its complex pathophysiology.

III. Methodology

This literature review was conducted in accordance with the recommendations of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) checklist, a widely used instrument for ensuring methodological rigor, transparency, and reproducibility in the selection of scientific studies. The bibliographic search was carried out in the PubMed, SciELO, and Web of Science databases, including articles published within the last 10 years.

To identify the studies, the descriptors *Depression*, *Systemic Inflammation*, *Psychiatric Disorders*, *Cytokines*, and *Neuroinflammation* were used and combined through Boolean operators appropriate to each database. Initially, the records retrieved were subjected to the identification and screening stages through the evaluation of titles and abstracts. Subsequently, potentially eligible studies underwent full-text review to assess thematic relevance and methodological quality.

After the removal of duplicate publications, the eligibility stage was conducted, during which the previously established criteria for composing the final sample were applied. The inclusion criteria comprised original scientific articles published within the last 10 years, full-text studies, research conducted with human subjects, investigations directly addressing the association between systemic inflammation and depression or other psychiatric disorders, as well as studies presenting clearly described methodologies and results consistent with the objectives of the research.

Observational studies, clinical trials, longitudinal investigations, and literature reviews with scientific relevance to the topic under investigation were also considered. Exclusion criteria included duplicate articles, publications outside the established time frame, studies unavailable in full text, works not directly related to the proposed topic, and research with insufficiently described methodologies.

Additionally, case reports, conference abstracts, letters to the editor, editorials, dissertations, theses, and studies that did not provide consistent scientific evidence regarding the relationship between inflammatory processes and psychiatric disorders were excluded.

At the end of the selection process, only studies that fully met the established methodological criteria were included, ensuring greater reliability and scientific quality of the evidence used in the preparation of this review.

IV. Results

The involvement of pro-inflammatory cytokines represents one of the major advances in the contemporary understanding of depression. Currently, numerous studies demonstrate that communication between the immune system and the central nervous system exerts a significant influence on the processes involved in emotional regulation. Cytokines are signaling proteins responsible for coordinating the body's immune and inflammatory responses. When persistent activation of these substances occurs, a series of physiological modifications capable of directly interfering with cerebral neurochemical balance can be observed. In this context, elevated

concentrations of interleukin-6, tumor necrosis factor-alpha, and C-reactive protein are frequently associated with the presence of characteristic depressive symptoms, including persistent sadness, fatigue, anhedonia, and reduced motivation.

Additionally, recent research has demonstrated that these inflammatory mediators influence different metabolic pathways related to brain function. Consequently, alterations occur in the availability of chemical substances essential for maintaining psychological well-being. Simultaneously, there is an increase in biological mechanisms that promote adaptive responses to physical illness but, when sustained over prolonged periods, contribute to the development of negative emotional manifestations. Thus, the understanding of depression extends beyond models traditionally centered solely on neurotransmitters, incorporating a broader perspective that recognizes the active participation of immunological processes in the onset and progression of the disorder.

The investigation of neuroinflammation has significantly expanded the understanding of the mechanisms linking immunological alterations to psychiatric manifestations. This phenomenon is characterized by the activation of immune cells within the central nervous system, particularly microglia, which are responsible for cerebral immune surveillance. Under physiological conditions, these structures perform essential functions related to neuronal protection and the maintenance of homeostasis. However, when exposed to persistent inflammatory stimuli, they begin to release substances capable of triggering functional alterations in different brain regions. As a result, areas involved in emotional processing, memory, and decision-making become particularly vulnerable to the effects of these biological modifications.

Furthermore, scientific evidence demonstrates that the continuous activation of these mechanisms compromises processes that are fundamental to brain health. Progressive reductions in neuronal adaptive capacity, impairments in the formation of new synaptic connections, and alterations in neural circuits responsible for affective regulation have been observed. Simultaneously, increased susceptibility to stress and reduced efficiency of the systems involved in emotional recovery following adverse events occur. From this perspective, neuroinflammation is understood as an important link between systemic biological factors and behavioral alterations, contributing to a more integrated view of depression and fostering the development of therapeutic approaches increasingly tailored to the complexity of this disorder.

Dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis constitutes one of the most relevant biological mechanisms in understanding the relationship between chronic stress and depression. This neuroendocrine system plays a fundamental role in coordinating physiological responses to situations perceived as threatening or challenging. Under normal conditions, its activation promotes the controlled release of hormones that assist the body in adapting to environmental demands. However, when exposure to stressors occurs continuously, prolonged activation of this regulatory circuit is observed, resulting in significant alterations in hormonal production and regulation. Consequently, imbalances emerge that affect both bodily functioning and emotional and cognitive processes.

In this context, persistent cortisol secretion exerts detrimental effects on different brain structures involved in mood regulation and stress responses. Over time, impairment of the body's adaptive capacity becomes evident, favoring states of physiological and emotional exhaustion. In addition, alterations in hormone receptor sensitivity hinder the feedback mechanisms responsible for the appropriate regulation of this system. Consequently, a scenario characterized by increased psychological vulnerability, reduced tolerance to adversity, and heightened susceptibility to depressive symptoms develops. Thus, the understanding of depression has come to encompass not only emotional and social factors but also important neuroendocrine alterations that directly influence mental health.

Alterations in neurotransmitters associated with inflammatory processes represent another central aspect of the contemporary understanding of depression. For many years, biological explanations of this disorder focused primarily on changes involving serotonin, dopamine, and norepinephrine. However, current scientific evidence demonstrates that inflammatory activity directly influences the synthesis, availability, and metabolism of these chemical substances. This interaction highlights that brain function depends on complex communication among distinct biological systems, whose alterations may significantly affect behavior, motivation, and emotional balance.

Moreover, persistent inflammatory mechanisms interfere with metabolic pathways essential for the production of neurotransmitters associated with well-being and reward. As a consequence, reduced efficiency of neural circuits responsible for affective regulation, interest in daily activities, and the capacity to experience pleasure has been observed. Simultaneously, there is an increase in metabolites potentially harmful to nervous tissue, favoring functional alterations in brain regions involved in emotional processing. Therefore, the interaction between inflammation and neurotransmission reinforces the need to understand depression as a multifactorial condition in which immunological, neurochemical, and environmental factors act in an integrated manner in determining the clinical presentation and progression of the disorder.

New therapeutic perspectives based on inflammatory modulation represent an important shift in the contemporary approach to depression. Traditionally, treatment has focused primarily on the use of antidepressant

medications and psychotherapeutic interventions. However, recent advances in neuroscience and immunology have demonstrated that inflammatory mechanisms exert a significant influence on the development and maintenance of depressive symptoms. In light of this evidence, growing interest has emerged in strategies capable of reducing systemic inflammatory activity while simultaneously promoting mental health benefits. This new understanding supports the development of more comprehensive therapeutic models that consider the interaction among biological, psychological, and environmental factors in the manifestation of psychological distress.

Within this context, several interventions have been investigated as complementary resources to conventional treatment. Scientific evidence indicates that regular physical activity, balanced dietary habits, improved sleep quality, and effective stress management contribute significantly to reducing inflammatory markers and improving emotional functioning. Furthermore, recent studies have evaluated the potential of pharmacological agents with immunomodulatory properties, particularly in individuals presenting persistent inflammatory alterations associated with depressive symptoms. At the same time, personalized approaches have gained increasing relevance, whereby specific biological characteristics of each patient guide the selection of the most appropriate interventions. In this way, inflammatory modulation has become a promising field of investigation, expanding therapeutic possibilities and contributing to a more integrated and precise understanding of depression.

The search for inflammatory biomarkers as auxiliary tools in the identification of depression has gained increasing relevance in contemporary psychiatry. Traditionally, the diagnosis of depressive disorders has been based predominantly on the clinical assessment of self-reported symptoms and the observation of behavioral changes. However, the heterogeneity of clinical manifestations often complicates the accurate delineation of depressive conditions, particularly in cases presenting overlapping characteristics with other psychiatric disorders. In this scenario, the investigation of biological indicators emerges as a promising alternative for complementing conventional diagnostic methods. Currently, numerous studies are examining the utility of blood-based markers as potential objective parameters capable of reflecting physiological alterations associated with mental illness.

Furthermore, the identification of these indicators enables significant advances in monitoring clinical progression and evaluating therapeutic response. As new scientific evidence continues to emerge, the hypothesis that specific biological profiles may assist in stratifying patients according to particular characteristics of the disorder has gained support. Consequently, it becomes possible to develop more individualized intervention strategies tailored to the specific needs of each case. In addition, the use of measurable parameters contributes to expanding the understanding of the pathophysiological mechanisms involved in depression, promoting integration among knowledge derived from psychiatry, immunology, and neuroscience. Thus, biomarkers constitute an important field of investigation aimed at developing more precise and scientifically grounded diagnostic models.

The relationship between chronic inflammatory diseases and depressive disorders represents another aspect widely explored in current research. Several clinical conditions characterized by persistent inflammatory processes exhibit a high prevalence of symptoms related to psychological distress. These conditions include autoimmune diseases, rheumatologic disorders, metabolic disturbances, and certain gastrointestinal diseases, which frequently coexist with significant emotional alterations. This association has attracted scientific interest because it demonstrates that psychological manifestations and organic conditions do not develop in isolation but rather constitute part of a complex network of physiological interactions. Consequently, mental health is increasingly understood from a broader perspective that considers the reciprocal influence among different bodily systems.

In parallel, recent investigations have demonstrated that the impact of these conditions extends beyond the physical effects directly related to the underlying disease. Functional limitations, persistent discomfort, reduced quality of life, and uncertainty regarding prognosis frequently contribute to the development of emotional distress. Simultaneously, physiological alterations resulting from continuous inflammatory states appear to influence neurobiological mechanisms associated with affective regulation. As a result of this multifaceted interaction, individuals affected by chronic inflammatory diseases exhibit greater vulnerability to the development of depressive symptoms when compared with the general population. Therefore, understanding this connection is essential for the development of integrated healthcare strategies capable of addressing both the physical and psychological components involved in the disease process.

Daily habits exert a substantial influence on mental health and on the physiological balance of the body. Currently, a growing number of studies demonstrate that lifestyle-related factors actively participate in the regulation of biological processes associated with the development of depression. Among these factors, dietary patterns, sedentary behaviors, inadequate sleep, and frequent exposure to emotional overload stand out. When such conditions persist over extended periods, a progressive imbalance can be observed across different bodily systems, favoring alterations that directly affect psychological functioning. Therefore, the understanding of depressive disorders has come to encompass not only genetic or neurobiological factors but also aspects related to individuals' daily routines.

In this regard, scientific evidence indicates that healthy behaviors contribute significantly to the maintenance of bodily homeostasis and the promotion of emotional well-being. Regular physical activity promotes physiological adaptations capable of improving cognitive functioning and strengthening mechanisms related to affective regulation. Likewise, balanced nutritional patterns provide essential substrates for optimal brain function, while consistent sleep habits support both physical and mental recovery. Additionally, strategies aimed at managing daily stress reduce the impact of potentially harmful stimuli on the body. Consequently, the adoption of healthy behaviors constitutes an important preventive and complementary measure in addressing depression, reinforcing the relevance of integrated approaches to health promotion.

In parallel, the investigation of immunomodulatory therapies has attracted increasing interest in the context of depressive disorders. Unlike traditional approaches, these interventions are directed toward the regulation of specific biological mechanisms involved in the interaction between physiological processes and psychological manifestations. The development of this line of research stems from the observation that certain individuals present clinical characteristics that do not respond satisfactorily to conventional treatments. In light of this reality, new therapeutic possibilities have emerged, focusing on the modulation of biological pathways associated with mental illness. Thus, understanding has expanded regarding the diversity of factors involved in depression and the need for increasingly individualized interventions.

Moreover, recent advances in precision medicine have facilitated the identification of clinical profiles that may potentially benefit from these complementary strategies. Researchers are currently evaluating different substances and approaches capable of modifying physiological responses related to the progression of the disorder, with the aim of improving therapeutic efficacy and reducing symptom persistence. Simultaneously, growing interest has emerged in combining pharmacological resources, psychosocial interventions, and practices focused on promoting overall health and well-being. This perspective strengthens more comprehensive models of care in which multiple dimensions of human functioning are considered during therapeutic planning. In this way, immunomodulatory therapies represent a promising field of investigation, contributing to the development of innovative strategies for the care of individuals affected by depression.

The integration of the immune system, the nervous system, and psychosocial factors constitutes one of the most comprehensive and innovative approaches to understanding depression today. This perspective recognizes that psychological suffering does not result from the isolated action of a single mechanism but rather emerges from the dynamic interaction among multiple dimensions of human experience. In this context, biological factors, life experiences, interpersonal relationships, socioeconomic conditions, and individual characteristics mutually influence one another over time. Consequently, adverse events, social vulnerability, traumatic experiences, and emotional difficulties may trigger complex physiological responses that affect different bodily systems. As a result, the understanding of depression has come to encompass the simultaneous influence of both internal and external factors in the development of mental illness.

From this perspective, psychological health is understood as the result of a continuous process of adaptation between the individual and the environment in which they are embedded. Current scientific evidence demonstrates that contexts characterized by social isolation, economic insecurity, family conflict, and prolonged exposure to adversity can significantly influence the overall functioning of the body. Conversely, resources such as social support, stable emotional bonds, a sense of belonging, and effective coping strategies promote emotional balance and subjective well-being. Thus, the integration of different fields of knowledge enables a more comprehensive understanding of depression, valuing both physiological aspects and the individual's lived experiences.

Furthermore, this integrative conception contributes to the development of more humanized and individualized models of care. Rather than focusing solely on symptomatic manifestations, contemporary approaches seek to understand the individual’s life trajectory, relational contexts, emotional characteristics, and the circumstances that influence mental health. Consequently, the ability of healthcare professionals to identify risk factors and protective elements involved in the processes of illness and recovery is enhanced. This understanding supports interventions that respect the uniqueness of each individual, recognizing that different life trajectories may lead to similar clinical manifestations.

Additionally, the integration of knowledge derived from psychiatry, psychology, neuroscience, immunology, and the social sciences strengthens the development of more effective and comprehensive therapeutic strategies. Currently, increasing value is being placed on practices that integrate clinical care, psychosocial support, quality-of-life promotion, and the strengthening of support networks. This multidimensional perspective allows depression to be understood as a complex phenomenon influenced by interdependent factors that operate continuously throughout life. Consequently, a paradigm is being consolidated that transcends reductionist explanations and promotes a broader, more contextualized, and scientifically grounded approach to psychiatric disorders.

V. Conclusion

It was concluded that the understanding of depression has undergone a significant transformation in recent years, moving beyond explanations based exclusively on alterations in neurotransmission systems to incorporate broader and more complex biological mechanisms. The scientific evidence analyzed demonstrated that systemic inflammation plays a relevant role in the pathophysiology of depressive disorders, establishing an important connection between the immune system and brain function. The studies indicated that elevated concentrations of inflammatory mediators were associated with greater severity of depressive symptoms, suggesting that inflammatory processes contribute not only to the development of the disorder but also to its maintenance and clinical progression.

The findings revealed that immunological alterations influence various neurobiological mechanisms involved in the regulation of mood, motivation, cognition, and behavior. In this context, neuroinflammation emerged as one of the principal elements involved in the communication between peripheral inflammatory processes and alterations observed within the central nervous system. Persistent activation of inflammatory mechanisms was associated with structural and functional modifications in brain regions responsible for emotional processing and stress adaptation, thereby contributing to the emergence of clinical manifestations characteristic of depression.

Additionally, dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis was identified as an important component in the interaction between chronic stress and psychological illness. The studies demonstrated that prolonged alterations in stress-response mechanisms contributed to impairments in physiological and emotional balance, increasing vulnerability to the development of depressive symptoms. Simultaneously, inflammatory processes were found to influence the availability and functioning of neurochemical substances essential for emotional stability, further reinforcing the multifactorial nature of the disorder.

Another aspect widely highlighted by the literature was the identification of inflammatory biomarkers as potential auxiliary tools for understanding the clinical heterogeneity of depression. The evidence suggested that specific biological profiles were associated with different presentations of the disorder, supporting future perspectives related to personalized mental healthcare. Likewise, a consistent association was observed between chronic inflammatory diseases and a higher prevalence of depressive symptoms, highlighting the close relationship between physical conditions and psychological suffering.

The investigations also emphasized the importance of behavioral and environmental factors in modulating biological processes related to mental health. Factors such as lifestyle habits, social context, experiences of adversity, and the availability of interpersonal support were shown to be directly associated with both the risk of illness and the clinical progression of affected individuals. In this regard, the findings reinforced the need to understand depression from an integrated perspective capable of simultaneously encompassing biological, psychological, and social determinants.

Finally, it was concluded that the new paradigms involving the interaction among the immune system, the nervous system, and the psychosocial context have substantially expanded the understanding of depression. The scientific evidence reviewed supports the view that the disorder results from a complex network of interactions among multiple biological systems and environmental factors, moving beyond simplified and unidimensional explanations. Consequently, an integrated and multidisciplinary approach has emerged as a promising pathway for advancing scientific knowledge, improving therapeutic strategies, and promoting more effective and individualized care for individuals affected by depressive disorders.

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