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Immunoglobulin E and Psychiatric Disorders: An Integrative Review

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Abstract: Immunoglobulin E (IgE) plays a key role in triggering allergic inflammatory responses and has traditionally been linked to conditions such as asthma, rhinitis, atopic dermatitis, and parasitic infections. However, growing evidence suggests that IgE may also be involved in psychiatric comorbidities, including anxiety, depression, sleep disorders, and stress-related syndromes. This article aims to provide an integrated view of the neuroimmune connections between IgE-mediated pathways and mental health. A narrative literature review was conducted by researching PubMed, Scopus, and Web of Science for studies published from 1999 to 2025, focusing on immunological, psychiatric, and psychosocial findings. The results of this synthesis show that IgE-mediated responses involve mast cells, eosinophils, and cytokines, which not only sustain allergic inflammation but also interact with the hypothalamic–pituitary–adrenal (HPA) axis and neurotrophic factors such as BDNF and NGF.

Keywords: Allergy, Anxiety, Depression, Inflammation, Neuroimmunology.

Review Paper

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1. INTRODUCTION

The blood is composed of cells and plasma, with leukocytes playing a central role in immune defense. Among them, monocytes differentiate into macrophages and dendritic cells, both specialized in phagocytosis and T antigen presentation to T lymphocytes, which is essential for adaptive immunity (Abbas *et al.*, 2000; Male *et al.*, 2006).

Lymphocytes are classified into B cells, responsible for antibody production, T cells, which regulate cell-mediated immunity, and natural killer (NK) cells, which act in innate defense against infected and tumor cells. Together, they form the cornerstone of immune surveillance and long-term memory (Table 1) (Chaplin, 2010; Delves *et al.*, 2017).

Table 1: Leukocytes are immune cells divided into several main types: neutrophils, which perform phagocytosis and destroy pathogens; lymphocytes (T, B, and NK cells), responsible for adaptive immunity and immune memory; monocytes/macrophages, which phagocytose and present antigens; eosinophils, that combat parasites and modulate allergic inflammation; and basophils, which release histamine and mediate hypersensitivity reactions

Leukocyte	Main Functions
Monocytes	Differentiate into macrophages and dendritic cells; phagocytosis; antigen presentation to T lymphocytes.
Lymphocytes	B cells: antibody production. T cells: cell-mediated immunity. NK cells: innate cytotoxic defense.
Neutrophils	First responders: Phagocytose bacteria/fungi; release enzymes and ROS.
Eosinophils	Defense against parasites; regulate allergic inflammation; immune modulation.
Basophils	Release histamine and mediators in allergy; support defense against parasites.

Other leukocytes, such as neutrophils, eosinophils, and basophils, are equally important. Neutrophils act as first responders, eliminating bacteria and fungi through phagocytosis and enzyme release. Eosinophils are involved in defense against parasites and in the regulation of allergic inflammation. Basophils, although less abundant, release histamine and other mediators that amplify inflammatory and allergic responses (Janeway *et al.*, 2001; Kumar *et al.*, 2015).

Complementing cellular defense, B lymphocytes produce immunoglobulins, which are classified into IgG, IgA, IgM, IgE, and IgD. Each class has distinct roles, ranging from long-term systemic

protection (IgG) to mucosal defense (IgA), initial immune responses (IgM), allergic reactions (IgE), and B cell receptor function (IgD). Together, they provide the humoral basis of immunity (Roitt *et al.*, 2017; Abbas *et al.*, 2021).

Table 2: Immunoglobulins (Ig) are antibody classes with distinct immune roles. IgG provides long-term defense and crosses the placenta; IgA protects mucosal surfaces; IgM is the first antibody produced in primary responses; IgE mediates allergic and parasitic responses via mast-cell activation; and IgD participates in B-cell receptor signaling and activation

Immunoglobulin	Main Functions
IgG	Long-term systemic protection; opsonization; complement activation; placental transfer to the fetus.
IgA	Mucosal defense in respiratory, gastrointestinal, and urogenital tracts; protection of newborns via breast milk.
IgM	The first antibody produced in the immune response; efficient in complement activation.
IgE	Mediates allergic reactions; Defends against helminths and other parasites.
IgD	Present on immature B cells; functions as an antigen receptor.

1.1 Objective

This study aims to analyze the interactions between immune mechanisms, psychosocial factors, and allergic conditions by reviewing scientific evidence from different disciplines.

2. METHODS

The present study adopted an integrative literature review as the methodological approach, aiming to gather and interpret scientific evidence related to the proposed theme. Initially, the research question was defined to guide the selection process and ensure the relevance of the studies. The inclusion criteria considered complete articles published in Portuguese, English, and Spanish, while exclusion criteria eliminated duplicated studies and those that did not align with the research objectives. Selected publications were examined for methodological consistency, thematic adequacy, and relevance to the research scope. Data analysis involved extracting essential information, such as study design, objectives, population, and principal findings. After classification, the evidence was synthesized in a narrative form, emphasizing convergences, divergences, and potential contributions to the scientific discussion. This methodological process enabled a comprehensive and critical understanding of the topic, providing a structured basis for interpreting the results and informing future directions.

3.0. STUDY SELECTION

3.1. Immunoglobulin E (IgE)

Plays a fundamental role in allergic inflammatory processes. It is a thermostable immunoglobulin of approximately 190 Kd that does not activate complement via the classical pathway, has an average serum half-life of five days, and does not cross the placenta (Linden *et al.*, 1999; Abbas *et al.*, 2000; O'Byrne, 2001; Peixoto, 2004). Normally present at low concentrations in the blood, IgE is bound to mast cells and basophils, contributing to allergic reactions and parasitic defense (Figure 1) (Arosa *et al.*, 2012; Lemos, 2023).

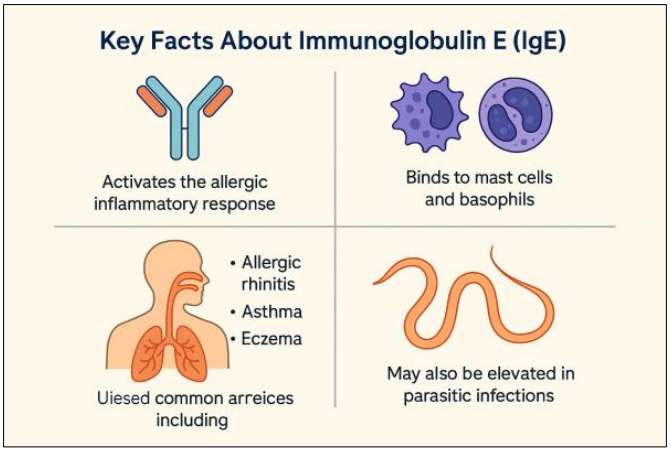

Figure 1: Immunoglobulin E (IgE) is a thermolabile antibody with a short serum half-life, primarily bound to mast cells and basophils. It plays a central role in allergic responses and is also elevated in parasitic infections and certain chronic conditions. Despite its low normal concentration in blood, IgE is crucial for hypersensitivity reactions and is closely associated with asthma, rhinitis, and atopic dermatitis
Source: Abbas *et al.*, (2000); Linden *et al.* (1999); O'Byrne (2001); Peixoto (2004)

Beyond its immunological role, growing evidence suggests that IgE and allergic diseases may influence psychological health, with links to mood disorders, anxiety, and stress-related conditions (Arosa *et al.*, 2012; Lemos, 2023; Immune Deficiency Foundation, 2024).

3.2. IgE in Allergic and Inflammatory Disorders

Elevated IgE levels are most commonly associated with allergic rhinitis, asthma, atopic dermatitis (also known as atopic eczema), parasitic infections, and inflammatory diseases such as Kawasaki disease and inflammatory bowel disease (Figure 2) (Lemos, 2023).

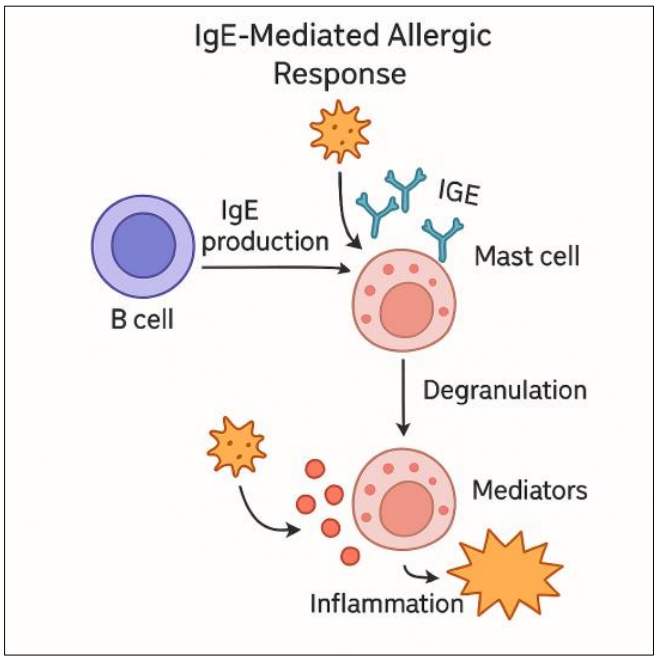

Figure 2: This diagram emphasizes the bidirectional interaction between the immune and nervous systems. Stress and psychological factors can affect immune responses, while allergic inflammation and immune dysregulation may impact brain function and mental health. Neuroimmune communication plays a key role in allergic diseases and psychiatric comorbidities, highlighting the importance of an integrative approach
Sources: Abbas *et al.*, 2000; Miller and Raison, 2016; Xu *et al.*, 2025

Hyper-IgE syndrome, a rare primary immunodeficiency, illustrates the systemic impact of dysregulated IgE, leading to recurrent infections, eczema, and skeletal abnormalities. The allergic inflammatory cascade involves mast cells, eosinophils, macrophages, and Th2 cytokines, which orchestrate IgE production and amplify immune responses (Linden *et al.*, 1999; Abbas *et al.*, 2000; Williamson and Snyder, 2013).

3.3. Psychiatric Comorbidities in IgE Mediated Conditions

Patients with allergic diseases frequently present with psychiatric comorbidities. Lichen Simplex Chronicus (LSC), for example, has been associated with depressive, anxiety, obsessive-compulsive symptoms, as well as sleep disturbances, sexual dysfunction, immune dysregulation, and mental health. Large population studies confirm higher rates of depression and anxiety in asthma, allergic rhinitis, and atopic dermatitis. Psychological stress may exacerbate allergic symptoms, while chronic inflammation worsens psychiatric outcomes, creating a bidirectional cycle. This underscores how allergic and inflammatory skin conditions often overlap with psychiatric comorbidities. (Figure 3) (Konuk *et al.*, 2007; Martín-Brufau *et al.*, 2010; Liao *et al.*, 2014; Kouris *et al.*, 2016; Altunaya *et al.*, 2021).

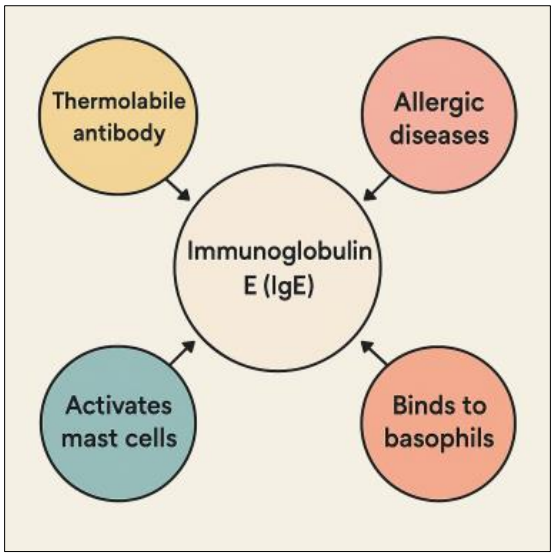

Figure 3: The immune system plays a dual role in health and disease. While protective responses mediated by IgE are essential in defending against pathogens, dysregulation can trigger allergies, chronic inflammation, and psychological impacts. Neuroimmune interactions further reveal how stress and emotional states modulate immune activity, linking dermatological and psychiatric conditions. Recent studies highlight the gut–brain–immune axis as a promising field for novel therapeutic approaches, reinforcing the importance of integrative perspectives
Source: Abbas *et al.* (2000); Miller and Raison (2016).

3.4. Mechanistic Pathways Linking IgE and Psychiatric Disorders

Mast cells release histamine, tryptase, and cytokines upon IgE cross-linking. These mediators influence the central nervous system by modulating microglia and astrocyte activity, contributing to neuroinflammation linked to depression and anxiety. Chronic allergic inflammation interacts with stress responses by activating the Hypothalamic–Pituitary–Adrenal (HPA) axis. Dysregulated cortisol rhythms have been observed in allergic patients with comorbid depression and anxiety (Figure 4) (Raap *et al.*, 2005; Papoiu, 2011; Rössing *et al.*, 2011).

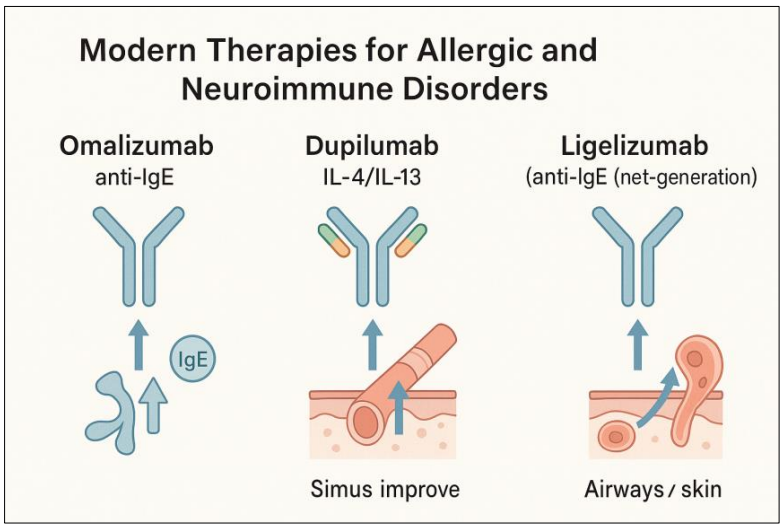

Figure 4: Modern biologic therapies modulate key pathways that drive allergic and neuroimmune disease. Omalizumab neutralizes circulating IgE and reduces downstream mast-cell activation; Dupilumab blocks IL-4/IL-13 signaling to dampen type-2 inflammation and improve airway and skin outcomes; Ligelizumab, a next-generation anti-IgE, enhances IgE blockade and may offer greater efficacy in selected patients
Source: Chipps *et al.*, 2012; Maurer *et al.*, 2013

Increased Nerve Growth Factor (NGF) and Altered Brain-Derived Neurotrophic Factor (BDNF) levels are found in allergic diseases and psychiatric disorders, suggesting shared neuroimmune mechanisms. Histamine and proteases from mast cells compromise blood–brain barrier integrity, enabling peripheral cytokines to drive neuroinflammatory cascades. Dysbiosis in allergic patients may alter IgE regulation

and contribute to psychiatric vulnerability through both immune and vagal pathways. Stress enhances IgE-mediated immune responses while IgE-driven inflammation exacerbates psychiatric symptoms, perpetuating a vicious cycle (Kiecolt-Glaser *et al.*, 2009; Papoiu, 2011; Erpolat *et al.*, 2017; He *et al.*, 2019; Sorour *et al.*, 2019; Xu *et al.*, 2025).

Accumulating clinical and experimental evidence supports a robust bidirectional relationship between IgE-mediated immune activation and psychiatric disorders. Patients with asthma, allergic rhinitis, or atopic dermatitis consistently show higher rates of depression, anxiety, and sleep disturbances compared with non-allergic populations, suggesting that IgE-driven inflammation extends beyond classical allergic symptoms to affect emotional and cognitive domains. Mast-cell degranulation triggered by IgE cross-linking releases histamine, tryptase, and cytokines that act on microglia and astrocytes, promoting neuroinflammation and altering synaptic plasticity (Table 3) (Raap *et al.*, 2005; Papoiu, 2011; Liao *et al.*, 2014; Kouris *et al.*, 2016).

Table 3: the main pathways linking IgE to psychiatric disorders, including immune activation, neuroinflammation, cortisol imbalance, neurotrophic alterations, and gut–brain interactions that contribute to mood and anxiety symptoms

Mechanism/Marker	Psychiatric Association	Supporting Pathway
Elevated total IgE	Increased risk of depression and anxiety in atopic patients	Immune activation and neuroinflammation
Mast cell degranulation (histamine, tryptase)	Sleep disturbance, mood changes	Microglial activation, blood–brain barrier alteration
Dysregulated cortisol rhythms	Anxiety, depressive symptoms	HPA axis disruption
Altered BDNF/NGF levels	Impaired neuroplasticity, cognitive symptoms	Neurotrophic imbalance
Gut microbiota dysbiosis	Stress vulnerability, mood instability	Gut–brain–immune axis

Dysregulation of the Hypothalamic–Pituitary–Adrenal (HPA) axis, with abnormal cortisol rhythms, has been observed in allergic patients presenting comorbid anxiety or depressive symptoms. Moreover, altered levels of neurotrophic factors such as BDNF and NGF, both critical for neuronal survival and connectivity, provide a mechanistic link between IgE-related inflammation and changes in mood and cognition. These converging pathways, immune activation, neurotrophin imbalance, and stress-axis dysregulation underscore the need for integrated therapeutic approaches that target both allergic and psychiatric manifestations (Figure 5) (Erpolat *et al.*, 2017; He *et al.*, 2019; Sorour *et al.*, 2019).

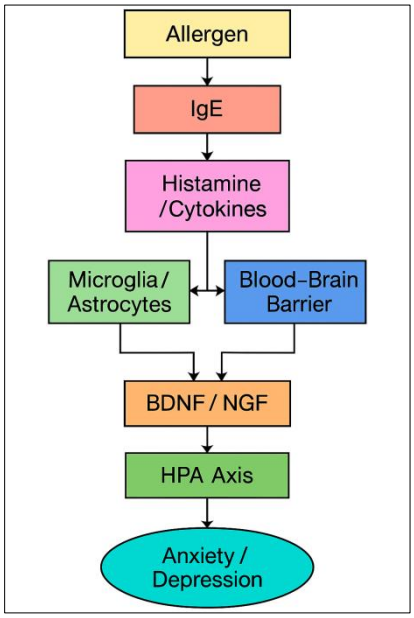

Figure 5: The main biological pathway linking allergen exposure to psychiatric symptoms. Allergens trigger IgE production, leading to mast cell activation and the release of histamine and cytokines. These mediators affect microglia, astrocytes, the blood–brain barrier, and neurotrophic factors such as BDNF and NGF, while also disrupting the HPA axis. Together, these processes promote neuroinflammation and increase the risk of anxiety and depression.
Sources: Raap *et al.* (2005) and Xu *et al.* (2025)

3.5. Genetic and Epigenetic Insights

Emerging evidence indicates that environmental and neuroimmune factors described in allergic diseases may exert their effects on IgE pathways through epigenetic mechanisms. Chronic stress, as well as microbiota dysbiosis, can influence gene expression without altering DNA sequences, creating long-lasting changes in immune regulation. The activation of the HPA axis and the release of cortisol, already documented in allergic patients with psychiatric comorbidities, may modify chromatin accessibility and the transcription of cytokines involved in IgE synthesis (Raap *et al.,* 2005; Miller and Raison, 2016; Xu *et al.,* 2025).

Likewise, altered levels of neurotrophic factors such as BDNF and NGF, observed in both allergic and psychiatric disorders, are susceptible to epigenetic control, providing a plausible link between stress responses, IgE production, and neuroplasticity (He *et al.*, 2019; Sorour *et al.*, 2019).

3.6. Biomarkers and Clinical Correlates

Several immunological and neuroendocrine markers described in allergic disorders may serve as potential indicators of psychiatric vulnerability. Total and allergen-specific IgE levels reflect the intensity of type-2 immune responses and have been associated with anxiety and depressive symptoms in patients with atopic diseases. Circulating cytokines produced by Th2 lymphocytes, such as interleukin-4 and interleukin-13, interact with the HPA axis, where dysregulated cortisol rhythms can influence both allergic inflammation and mood regulation (Raap *et al.*, 2005; Rössing *et al.*, 2011; Miller and Raison, 2016).

Neurotrophic factors, including BDNF and NGF, show altered levels in allergic and psychiatric conditions and are critical for neuronal plasticity. In addition, gut microbiota composition influences immune homeostasis and IgE production, providing another layer of biomarker potential through microbiota-derived metabolites and their impact on stress responses. Although these markers are not specific to a single psychiatric diagnosis, their combined evaluation may help identify patients at higher risk for mood and anxiety disorders and guide therapeutic strategies that target both immune and neuroendocrine pathways (Erpolat *et al.,* 2017; He *et al.,* 2019; Sorour *et al.,* 2019; Fiocruz-ARCA, 2024).

3.7. Environmental and Lifestyle Modulators

Environmental exposures and lifestyle factors play a key role in shaping IgE responses and their psychiatric links. Chronic psychosocial stress, urban pollution, and diets high in ultra-processed foods have all been shown to boost type-2 immune activation and disrupt the HPA axis, thereby promoting both allergic inflammation and mood issues. On the other hand, protective factors like regular physical activity, balanced diets rich in fruits, vegetables, and omega-3 fatty acids, and enough sleep help maintain immune balance and may lower circulating IgE levels (Miller and Raison, 2016; UNICAMP-FCM, 2025; USP Digital, 2025; Xu *et al.,* 2025).

These habits also improve microbiota diversity, supporting gut–brain signaling pathways that buffer stress responses and enhance emotional regulation. Although lifestyle changes are not disease-specific, their ability to influence immune mediators, neuroendocrine rhythms, and mental health at the same time underscores their value as complementary strategies in managing IgE-mediated conditions and psychiatric comorbidities (Fiocruz-ARCA, 2024; USP-FMUSP, 2025a; Xu *et al.,* 2025).

3.8. Therapeutic Approaches and Clinical Perspectives

Omalizumab reduces free IgE, controlling asthma and urticaria while also improving anxiety and depression symptoms. Newer drugs such as ligelizumab offer enhanced efficacy. Dupilumab, targeting IL-4Rα, improves both allergic symptoms and psychological symptoms. l well-being, particularly in atopic dermatitis. SSRIs and other psychotropics may benefit allergic patients with psychiatric symptoms, but careful monitoring of immune interactions is required (Figure 5) (Chida *et al.,* 2008; Miller and Raison, 2016; Simpson *et al.,* 2016; Zeller *et al.,* 2021).

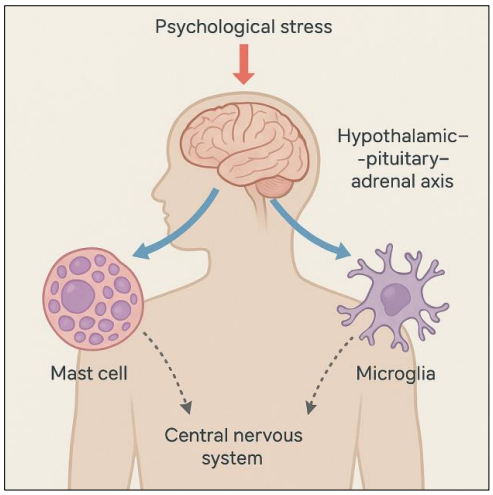

Figure 6: This figure illustrates the interaction between psychological stress, the central nervous system, mast cells, and microglia. Stress signals are mediated through the hypothalamic–pituitary–adrenal axis and autonomic nervous system, influencing immune and neuroinflammatory responses
Source: Scientific literature on neuroimmunology and stress-related mechanisms (2025)

Cognitive-behavioral therapy and mindfulness interventions reduce psychiatric symptoms and may indirectly alleviate allergic flares. Dietary changes, probiotics, and physical activity modulate immune and psychiatric outcomes simultaneously. Biomarker research is advancing to identify patients at risk of both allergic and psychiatric comorbidities. Given the high prevalence of allergies worldwide (25–30%), integrating allergy and psychiatry in public health strategies is crucial (Chipps *et al.*, 2012; Maurer *et al.*, 2013; PneumoCenter, 2023; USP-FMUSP, 2025b).

3.9. Future Directions

Emerging fields such as epigenetics, microbiome modulation, and digital health tools promise to advance the understanding of the IgE-psychiatry link. Precision medicine integrating immune and psychiatric profiling may redefine prevention and treatment strategies (Figure 7) (Xu *et al.*, 2025).

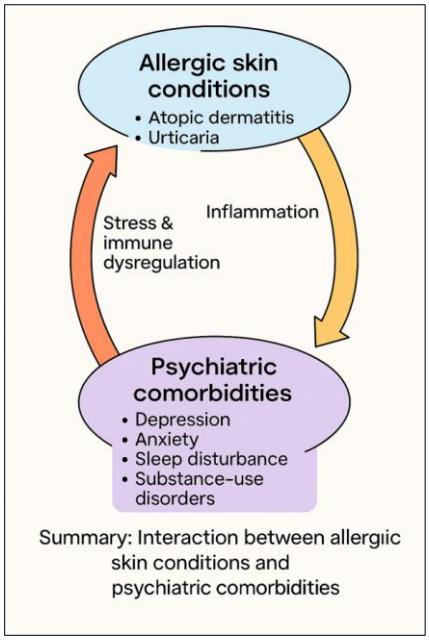

Figure 7: Neuroimmune Signaling in IgE-Mediated Disorders. This diagram highlights the flow of immune communication in IgE-mediated conditions. Allergens activate IgE antibodies bound to mast cells and basophils, triggering the release of histamine and inflammatory mediators. These mediators interact with nerve endings, amplifying itch and stress responses, while neuropeptides influence immune cells, creating a bidirectional neuroimmune loop that sustains chronic symptoms
Source: Abbas *et al.* (2000); Raap *et al.*, (2005)

3.7. Neuroimmune Pathways and IgE-Mediated Mechanisms

The interaction between the immune and nervous systems has received increasing attention in recent years, especially regarding allergic and inflammatory conditions. Immunoglobulin E (IgE) plays a crucial role in these processes, serving as a key mediator of hypersensitivity reactions. When IgE binds to high-affinity receptors (FcεRI) on mast cells and basophils, subsequent allergen exposure causes degranulation and the release of histamine, cytokines, and neuropeptides. This cascade not only triggers classic allergic responses such as pruritus and wheezing but also contributes to neuroimmune signaling that may affect psychiatric comorbidities like anxiety and depression (Raap *et al.*, 2005; Papoiu, 2011; Erpolat *et al.*, 2017; Xu *et al.*, 2025).

IgE, once considered exclusively an allergy mediator, is increasingly recognized as a bridge between immunology and psychiatry. Elevated IgE levels and allergic inflammation are strongly associated with psychiatric morbidity, mediated by neuroinflammation, HPA dysregulation, neurotrophins, and gut–brain interactions. Current therapies targeting IgE and cytokines demonstrate dual benefits for physical and psychological health. Future research on biomarkers, microbiota, and precision medicine will enable integrated and holistic care. Recognizing the interplay between IgE and psychiatric disorders is a critical step toward improving global health and well-being (Raap *et al.*, 2005; Papoiu, 2011; Erpolat *et al.*, 2017; Xu *et al.*, 2025).

Emerging evidence suggests that the IgE-mediated pathway does not operate in isolation but rather interacts with the hypothalamic-pituitary-adrenal (HPA) axis and stress-related mechanisms. Altered cortisol rhythms, for example, can amplify inflammation and modulate neuronal plasticity, creating a bidirectional relationship between immune responses and mental health. This integrative framework helps explain why patients with chronic allergic diseases, such as atopic dermatitis or urticaria, frequently report psychiatric symptoms, and why biologic treatments targeting IgE or interleukin pathways often improve both dermatological and psychological outcomes (Maurer *et al.*, 2013; Kouris *et al.*, 2016; Journal da USP, 2017; Fiocruz-IFF, 2022; Zeller *et al.*, 2021).

Epidemiological data reinforce this relationship: according to the World Health Organization, 25% of the global population suffers from some form of allergy, reaching 30% in Brazil. Beyond physical symptoms, allergies impose dietary, environmental, and lifestyle restrictions, fueling anxiety, social isolation, depression, and reduced self-esteem. Thus, IgE-mediated diseases exert both immunological and psychological burdens, creating a two-way interaction between allergy and mental health (Daher *et al.*, 2009; PneumoCenter, 2023).

3.10. Limitations of the Study

This study has some limitations that should be recognized. First, the integrative literature review depends on the availability and quality of existing studies, which may introduce publication bias and limit the scope of evidence. Second, although articles were included in multiple languages and from various databases, relevant research outside these criteria might have been missed. Third, the synthesis was based on secondary data without direct experimental validation, restricting the ability to establish causal relationships. Lastly, rapid scientific advances in immunology and neuroimmune interactions could make some findings outdated over time, emphasizing the importance of ongoing updates in future reviews (Kouris *et al.*, 2016; Zeller *et al.*, 2021).

4.0. CONCLUSION

This study highlights the complex interplay between IgE-mediated immune responses, neuroimmune signaling, and their psychosocial implications. The evidence suggests that allergic diseases are not limited to physiological mechanisms but also extend to mental health, emphasizing the role of stress, neurotrophins, and inflammatory pathways. Advances in targeted therapies, such as monoclonal antibodies, offer promising outcomes, yet challenges remain in fully addressing the multifactorial nature of these conditions. By integrating immunological, neurological, and psychological perspectives, this review underscores the importance of interdisciplinary approaches for more effective diagnosis, treatment, and patient quality of life.

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