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ALLERGIC RHINITIS AND MENTAL HEALTH: EPIDEMIOLOGICAL ASSOCIATIONS AND NEUROIMMUNE MECHANISMS

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ABSTRACT

Allergic rhinitis (AR) is a common chronic condition associated with significant quality-of-life impairment. In recent years, increasing attention has been directed toward the potential relationship between AR and mental health outcomes. Epidemiological studies have reported associations between AR and a range of psychological conditions, including depression, anxiety, sleep disturbances, and impaired cognitive performance.

Emerging evidence from psychoneuroimmunology provides several potential mechanisms that may explain these relationships. Peripheral allergic inflammation may influence central nervous system function through cytokine-mediated immune–brain communication, histaminergic signaling, alterations in blood–brain barrier permeability, and neuroendocrine responses related to stress. In addition, sleep disturbances caused by nasal obstruction and inflammatory mediators may represent an important intermediary pathway linking allergic symptoms with mood and cognitive changes.

This narrative review summarizes current evidence on the epidemiological associations between allergic rhinitis and mental health outcomes and discusses potential neuroimmune mechanisms underlying these interactions. Understanding these complex relationships may contribute to a more comprehensive approach to the management of allergic rhinitis and highlight the importance of considering psychological and sleep-related factors in affected patients.

KEYWORDS

Allergic Rhinitis, Mental Health, Depression, Anxiety, Neuroimmune Interactions, Sleep Disturbances

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Introduction

Allergic rhinitis (AR) is a highly prevalent chronic inflammatory condition affecting approximately 10–20% of the population in the United States and up to 50% worldwide (Mims, 2014; Wise et al., 2023). Its prevalence has increased steadily over time, particularly in industrialized and urbanized regions, reflecting a growing global health burden. (Elholm et al., 2016; Licari et al., 2023). AR frequently begins in childhood or adolescence and often persists into adulthood, thereby affecting individuals during key educational and occupational periods of life (Greiner et al., 2011).

The cardinal symptoms of AR—rhinorrhea, nasal congestion, itching, and sneezing—are commonly perceived as mild and self-limiting (Nathan, 2007). However, accumulating evidence indicates that AR substantially impairs quality of life and imposes a considerable socioeconomic burden, particularly when inadequately diagnosed or treated (Bernstein et al., 2024; Dierick et al., 2020). The economic impact of AR comprises both direct and indirect costs. Direct costs include expenditures related to physician visits, pharmacotherapy, and other therapeutic interventions. Indirect costs, by contrast, arise from work absenteeism and reduced on-the-job productivity (presenteeism) (Stróżek et al., 2019). Notably, several studies suggest that in AR, indirect costs exceed direct healthcare expenditures. In a multicenter Spanish study conducted by Colás et al. (Colás et al., 2017), indirect costs were estimated at approximately €1,772.90 per patient-year, largely attributable to presenteeism. Similarly, a large survey-based study in the United States demonstrated that productivity losses associated with AR (approximately USD 593 per employee-year) exceeded those reported for other chronic conditions such as diabetes, asthma, or coronary heart disease (Lamb et al., 2006). These findings indicate that AR has substantial clinical and socioeconomic impact.

Such substantial productivity losses may be partially explained by the high prevalence of mental health disturbances among individuals with AR. A meta-analysis encompassing 49 studies (Safia et al., 2024) reported pooled prevalence rates of approximately 25% for depression, 25% for anxiety, 65% for stress, 14% for suicidal thoughts, 48% for poor sleep quality and 36% for insomnia among patients with AR.

Studies in psychoneuroimmunology show that systemic inflammation can alter emotional and cognitive processes through immune–brain interactions (Bower & Kuhlman, 2023). Low-grade systemic inflammation, elevated pro-inflammatory cytokines, and alterations in neuroimmune signaling have been increasingly implicated in the pathophysiology of mood disorders (Harsanyi et al., 2022). In this context, allergic inflammation may contribute to psychiatric vulnerability. Moreover, sleep disturbance, a hallmark feature of poorly controlled AR due to nocturnal nasal obstruction, may mediate the relationship between allergic symptoms and mental health impairment (Yang et al., 2024).

Although epidemiological studies consistently show associations between AR and mental health outcomes, key gaps remain, particularly regarding causal relationships and integrated mechanistic models.

The aim of this narrative review is to synthesize current evidence on the relationship between allergic rhinitis and mental health outcomes, with particular emphasis on epidemiological associations and underlying biological mechanisms.

Pathophysiology of Allergic Rhinitis

AR represents an example of a hypersensitivity reaction mediated by immunoglobulin E (IgE) (Dispenza, 2019). In sensitized individuals, exposure to environmental aeroallergens leads to disruption of the epithelial barrier, partly due to proteolytic enzymes contained within these allergens. This epithelial injury triggers the release of so-called “alarmins,” including interleukin (IL)-25, IL-33, and thymic stromal lymphopoietin (TSLP), as well as damage-associated molecular patterns (DAMPs). These mediators activate innate and adaptive immune pathways, promoting a type 2 immune response (Kucuksezer et al., 2023).

Activation of T helper 2 (Th2) lymphocytes and group 2 innate lymphoid cells (ILC2s) results in the production of characteristic type 2 cytokines, particularly IL-4, IL-5, and IL-13 (Scadding, 2014). IL-4 and IL-13 stimulate B cells to undergo class switching and produce allergen-specific IgE antibodies. These IgE molecules bind with high affinity to FcεRI receptors expressed on mast cells and basophils. Upon subsequent allergen exposure, cross-linking of IgE bound to FcεRI triggers rapid degranulation of these effector cells, leading to the release of preformed mediators such as histamine, tryptase, and leukotrienes. This immediate (early-phase) reaction is responsible for the hallmark symptoms of AR, including sneezing, rhinorrhea, pruritus, and nasal congestion (Eifan & Durham, 2016).

In many patients, an additional late-phase response develops approximately 4–8 hours after allergen exposure. This phase is characterized by the recruitment and activation of eosinophils, basophils, neutrophils, monocytes, and T lymphocytes (Dispenza, 2019; Wise et al., 2023). Structural cells of the nasal mucosa, including epithelial cells and fibroblasts, further amplify inflammation by secreting chemokines and adhesion molecules, thereby perpetuating cellular infiltration and tissue remodeling (J.-X. Li et al., 2026).

Clinically, allergic rhinitis may present either as seasonal allergic rhinitis (SAR) or perennial allergic rhinitis (PAR). SAR is typically triggered by exposure to pollens from trees, grasses, or weeds and occurs during specific periods of the year corresponding to pollen seasons. In contrast, PAR is usually associated with year-round exposure to indoor allergens such as house dust mites. Many patients may also experience a mixed pattern with both seasonal exacerbations and persistent baseline symptoms (Dykewicz et al., 2020).

In addition to this traditional classification, the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines propose a clinically oriented classification based on symptom duration and severity. According to ARIA, allergic rhinitis may be categorized as intermittent (symptoms present fewer than four days per week or fewer than four consecutive weeks) or persistent (symptoms present at least four days per week and for at least four consecutive weeks). Furthermore, disease severity is classified as mild or moderate-to-severe, depending on the degree of impairment in sleep, daily activities, work or school performance, and the overall burden of symptoms (Brozek et al., 2010).

Epidemiological Evidence Linking AR and Mental Health

Depression and anxiety

Numerous studies, including meta-analyses, cohort studies, and cross-sectional analyses, have reported an increased prevalence of depression and anxiety among individuals with AR (Lu et al., 2018; Safia et al., 2024) . One meta-analysis (J. Rodrigues et al., 2021) estimated that the risk of developing depressive symptoms in individuals with AR is approximately 1.6 times higher than in healthy controls, while the risk of anxiety is even higher, reaching approximately 1.9-fold. Consequently, nearly one-third of patients with AR may experience clinically relevant depressive symptoms during the course of the disease. Interestingly, some studies indicate that this association may be more pronounced in women than in men, suggesting a potential role of sex-related biological or psychosocial factors (Bedolla-Barajas et al., 2017; Lu et al., 2018).

However, the relationship between depressive or anxiety-related symptoms and AR severity remains inconsistent. Some studies suggest that individuals with poorly controlled or more severe AR exhibit higher depression and anxiety scores (Jorge Rodrigues et al., 2023), whereas others have not confirmed this association (Bedolla-Barajas et al., 2017). Notably, increased rates of depression and anxiety have also been observed in patients with both allergic and non-allergic rhinitis, raising the possibility that symptoms such as nasal obstruction, rather than the allergic process itself, may contribute to psychological burden. This interpretation is to some extent supported by findings linking primary olfactory dysfunction with increased prevalence of depressive symptoms (Kohli et al., 2016).

In addition, evidence from animal models suggests that allergen sensitization may induce behavioral changes resembling anxiety, accompanied by altered neuronal activity in brain regions involved in emotional regulation (Salimi et al., 2019). Experimental studies have also shown that transient anosmia may induce depressive-like behavior (Ahn et al., 2018).

Suicidal Ideation and Attempts

The relationship between allergic rhinitis and suicidal behavior has also been explored. A recent meta-analysis (Høj et al., 2025) reported no significant increase in the prevalence of suicidal ideation among individuals with AR, but a significant increase in both suicide attempts and suicide-related mortality.

Specifically, individuals with AR were approximately 25% more likely to attempt suicide and 65% more likely to die by suicide compared with individuals without AR. Interestingly, this increased risk was not observed in populations younger than 18 years of age. It should be noted, however, that the meta-analysis was based predominantly on cross-sectional studies, which limits the ability to determine causality of these associations. Interestingly, although the existence of spring peak in suicidal attempts is still poorly understood, it could potentially be associated with the seasonal peak of the aeroallergens (Postolache et al., 2008).

Sleep Disorders

Poor sleep quality, excessive daytime sleepiness, and chronic fatigue are common complaints among individuals with AR (Bernstein et al., 2024; Greiner et al., 2011). A meta-analysis (Liu et al., 2020) has demonstrated higher rates of insomnia, restless sleep, nocturnal awakenings, obstructive sleep apnea, snoring, and nocturnal enuresis in patients with AR compared with healthy controls. In addition, daytime consequences such as difficulty waking up and increased use of sleep medications are more frequently reported in this population.

Quality of Life

Reduced quality of life has been consistently demonstrated in studies utilizing standardized questionnaires designed to assess health-related quality of life and disease burden. The most commonly reported complaints include impaired physical, social, and emotional functioning, decreased energy levels, and a poorer overall perception of health (Meltzer, 2001; Small et al., 2013).

These findings are further supported by studies demonstrating seasonal changes in cognitive performance among individuals with seasonal AR. During periods of high allergen exposure, affected individuals tend to perform worse on objective tests assessing domains such as learning, memory, and decision-making. In addition, impairments in psychomotor performance and reductions in positive affect have been observed during allergy seasons compared with winter months (Meltzer, 2001).

The clinical significance of these observations is underscored by the considerable loss of productivity associated with AR (Dierick et al., 2020). Importantly, effective treatment of AR has been shown to significantly improve patient-reported quality of life and reduce the functional burden associated with the disease (Sousa-Pinto et al., 2025).

Pediatric Population

AR is highly prevalent among children, with self-reported prevalence estimates approaching 20% in many populations (Licari et al., 2023).

AR has long been recognized to negatively affect academic performance**.** Children with AR frequently report difficulties with concentration, increased fatigue, and daytime sleepiness, which may interfere with learning and academic achievement, as well as participation in school activities (Meltzer, 2001). These effects may be further exacerbated by sleep disturbances, which are commonly reported in pediatric AR patients (D'Elia et al., 2022)..

Additionally, a potential association between AR and neurodevelopmental disorders has been suggested. A recent meta-analysis (Wang et al., 2024) reported a significant correlation between AR and attention-deficit/hyperactivity disorder (ADHD), with children diagnosed with ADHD having approximately 1.84 times higher risk of developing AR compared with non-ADHD controls.

Comorbidities and potential confounding factors

Allergic rhinitis frequently coexists with other atopic and inflammatory conditions, most notably asthma, atopic dermatitis, conjunctivitis, chronic rhinosinusitis, tension headaches and migraine (Bernstein et al., 2024). Importantly, many of these have themselves been independently associated with an increased risk of mental health disturbances, including depression, anxiety, and sleep disorders (Conway et al., 2024).

The frequent coexistence of these conditions may therefore complicate the interpretation of epidemiological findings investigating the relationship between allergic rhinitis and mental health outcomes.

Neuroimmune Pathways Linking Allergic Rhinitis and Brain Function

Cytokine Signaling and Brain Function

Pro-inflammatory cytokines can modulate brain activity through several complementary pathways. Peripherally produced cytokines may activate afferent vagal nerve fibers, transmitting inflammatory signals to brainstem nuclei (Undem & Taylor-Clark, 2014). In the context of AR, it has been hypothesized that inflammatory mediators generated in the nasal mucosa may exert direct central effects due to the anatomical proximity of the olfactory epithelium to the brain. This proposed “nose–brain interface” suggests that inflammatory signals could access the central nervous system (CNS) via olfactory pathways or trigeminal afferents (Mou et al., 2024; Undem & Taylor-Clark, 2014). Animal studies have demonstrated that intranasal administration of aeroallergens or inflammatory stimuli can induce expression of pro-inflammatory cytokine genes in brain regions involved in emotional and cognitive processing (Tonelli & Postolache, 2010).

Alternatively, cytokines may reach the CNS via humoral routes, including active transport across the blood-brain barrier (BBB) or through circumventricular organs, where the barrier is more permeable (Erickson & Banks, 2018). Importantly, the inflammatory response in AR is not limited to the nasal mucosa and circulating levels of pro-inflammatory cytokines—such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α), or interleukin-6 (IL-6)—have been shown to increase during active allergic inflammation (Borish, 2003; Nur Husna et al., 2022).

These cytokines influence multiple neurobiological processes. They alter monoaminergic neurotransmission, including serotonin and dopamine metabolism; reduce hippocampal neurogenesis; impair synaptic plasticity; and modulate neural circuits critical for mood regulation and executive function (Bower & Kuhlman, 2023; Felger & Miller, 2012; Kappelmann et al., 2018). Collectively, these changes can give rise to “sickness behavior,” characterized by fatigue, reduced motivation, social withdrawal, impaired concentration, and depressed mood (Hennessy et al., 2014). Multiple studies have reported elevated circulating inflammatory markers, including TNF-α, IL-6, and C-reactive protein (CRP), in patients with major depressive disorder (Colasanto et al., 2020; Dowlati et al., 2010) Moreover, experimental induction of systemic inflammation, most commonly using lipopolysaccharide or vaccination models, has been shown to reproduce behavioral and affective symptoms resembling depressive states (Bower & Kuhlman, 2023). However, most experimental and clinical studies have focused on non-allergic inflammatory stimuli, and it remains unclear to what extent allergic inflammation, particularly Th2-type cytokines, elicit similar neuroimmune alterations.

Blood–Brain Barrier and Histaminergic Signaling

The BBB represents a critical regulator of the extent to which peripheral immune activation can influence CNS function. BBB permeability may be altered during inflammatory processes, potentially facilitating the entry of cytokines and other immune mediators into the brain. Endothelial dysfunction and disruption of tight junctions occurring during systemic inflammation may further amplify neuroimmune communication (Erickson & Banks, 2018).

Additionally, mast cells have been identified in close proximity to the BBB, where they may function as a relay between peripheral inflammatory signals and the brain (Yue et al., 2023). Upon activation, mast cells release a range of mediators, including pro-inflammatory cytokines, tryptase, and neurotransmitters such as serotonin and—most notably—histamine (Thangam et al., 2018).

Histamine signaling provides a potential mechanism linking allergic inflammation with neuropsychiatric symptoms. Histamine receptors are widely expressed throughout the CNS. The H3 receptor, in particular, functions as a presynaptic autoreceptor regulating histamine release and modulating the release of several other neurotransmitters, including serotonin, dopamine, and acetylcholine (Carthy & Ellender, 2021).

Histaminergic neurons originating from the tuberomammillary nucleus of the hypothalamus project widely throughout the brain and play a central role in the regulation of arousal, cognition, and circadian rhythms (Yoshikawa et al., 2021). Through these extensive projections, histamine influences multiple neural circuits involved in attention, wakefulness, and emotional regulation. This role is reflected clinically in the sedative and anxiolytic effects of first-generation antihistamines, which readily cross the BBB and antagonize central H1 receptors. Moreover, different agents within the same pharmacological class may exert distinct central effects, highlighting the complexity of histaminergic neurobiology (Isomura et al., 2014; Yanai et al., 2012).

H1 and H4 receptors are expressed on microglial cells, where experimental studies in murine models suggest that histamine signaling may contribute to neuroinflammatory processes, including phagocytosis and the generation of reactive oxygen species. These mechanisms have been implicated in dopaminergic neuron toxicity within the substantia nigra (Rocha et al., 2016). In addition, H1, H2, and H3 receptors have been identified on astrocytes, and histamine has been shown to regulate astrocytic release of neurotrophins and other neuromodulatory factors (Carthy & Ellender, 2021). Histamine has also been implicated in the regulation of endothelial function (Steelant et al., 2018), although its precise role in maintaining BBB integrity remains incompletely understood.

Proinflammatory cytokines released during allergic inflammation may further modulate histaminergic neurotransmission, suggesting an additional pathway linking peripheral immune activation with central nervous system function (Carthy & Ellender, 2021).

Consequently, histamine is thought to participate in multiple fundamental neurobiological processes, including neuronal excitability, synaptic transmission, regulation of neuroinflammation, brain metabolism, and neuronal plasticity. Through these mechanisms, histaminergic signaling may potentially contribute to the development or modulation of neurodevelopmental and neuropsychiatric disorders such as ADHD (Carthy & Ellender, 2021).

However, the role of histamine in brain function remains complex and incompletely understood. Most available studies have focused primarily on neuronal sources of histamine rather than mast cell-derived histamine. Furthermore, many investigations have been conducted in vitro or in animal models, often outside the context of allergic disease. As a result, the precise contribution of histaminergic signaling to CNS manifestations in allergic conditions remains insufficiently characterized and warrants further investigation.

Microglial Activation

Microglia, the resident immune cells of the CNS, represent a central mediator of neuroinflammation. They respond to peripheral immune signals and can amplify inflammatory cascades within the brain. Beyond their immune function, microglia are essential for synaptic pruning, a process critical for normal neurodevelopment and circuit refinement (Smail & Lenz, 2024).

Experimental studies suggest that cytokines central to allergic inflammation, such as IL-4, may influence microglial function. In animal models, postnatal elevations in IL-4 have been associated with altered microglial-mediated synaptic pruning (Guedes et al., 2023). Given that AR frequently affects children and adolescents, periods characterized by dynamic neurodevelopment, such immune-mediated alterations could theoretically influence emotional and cognitive maturation (Carthy & Ellender, 2021). However, direct evidence linking AR-associated cytokine profiles to microglial dysfunction in humans remains limited.

Neuropsychiatric Effects of AR Pharmacotherapy

Current therapeutic strategies aim to control symptoms and suppress allergic inflammation. Intranasal corticosteroids represent the first-line pharmacological treatment in persistent or moderate to severe AR. Antihistamines, administered either orally or intranasally, target histamine-mediated symptoms such as sneezing, itching, and rhinorrhea. Second- and third-generation oral antihistamines are generally preferred because of their improved safety profile. Additional pharmacological options include leukotriene receptor antagonists, such as montelukast, which are primarily recommended for patients with concomitant asthma. Finally, allergen immunotherapy remains the only disease-modifying treatment for allergic rhinitis, as it may provide sustained clinical benefits even after treatment discontinuation(Bernstein et al., 2024; Wise et al., 2023).

Pharmacological treatment of AR may independently influence mental health outcomes. First-generation antihistamines, although no longer recommended as first-line therapy, could still be used by some patients. Agents such as diphenhydramine readily penetrate the BBB, also exhibit antimuscarinic effect, and are associated with sedation, impaired attention and memory, and reduced psychomotor performance (Seidman et al., 2015). These effects may be particularly detrimental in children and adolescents, potentially affecting their academic performance.

In contrast, second- and third-generation antihistamines were developed to minimize central penetration and thereby reduce sedation and cognitive impairment. mild sedative effects have been reported with certain agents, suggesting that central histaminergic modulation cannot be entirely excluded. (Kawauchi et al., 2019; Mann et al., 2000).

Montelukast has been associated with neuropsychiatric adverse effects, including depression, anxiety, insomnia, nightmares, and suicidal ideation (Paljarvi et al., 2022). The use of synthetic glucocorticoid was associated in a recent meta-analysis (Koning et al., 2024)with higher incidence of mental health problems, in particular mood disorders, although it remains unclear to what extent the use of nasal formulations can be involved.

Stress, Neuroendocrine Regulation, and Allergic Inflammation

Studies in psychoneuroimmunology have elucidated mechanisms through which chronic stress may influence immune function and contribute to allergic inflammation (Marshall, 2020). Central to this process is activation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic–adrenal–medullary (SAM) system (Turner et al., 2020).

Persistent psychological stress leads to sustained secretion of glucocorticoids (primarily cortisol) and catecholamines (epinephrine and norepinephrine). These stress mediators exert complex immunomodulatory effects and appear to promote a shift from Th1- to Th2-dominant immune responses, favoring an allergic phenotype. Mechanistically, catecholamines and glucocorticoids suppress IL-12 production by antigen-presenting cells, thereby inhibiting Th1 differentiation. Concurrently, glucocorticoids may enhance the production of Th2-associated cytokines, including IL-4, IL-5, and IL-13, which are central to the pathogenesis of allergic diseases such as AR (Lira et al., 2022; Montoro et al., 2009).

Experimental studies have demonstrated increased expression of Th2-type cytokines in both healthy and atopic individuals exposed to acute stress paradigms (Assaf et al., 2017; Lira et al., 2022). However, the degree to which stress-induced immunological shifts translate into clinically significant exacerbation of allergic symptoms remains incompletely understood and likely depends on disease severity, chronicity, and individual susceptibility.

Importantly, chronic stress may paradoxically promote inflammation through the development of glucocorticoid receptor resistance (Hassamal, 2023). Sustained HPA activation can impair negative feedback regulation and reduce cellular responsiveness to cortisol. As a result, despite elevated circulating glucocorticoid levels, anti-inflammatory signaling may become ineffective, contributing to a pro-inflammatory milieu. This mechanism has been implicated in both allergic and psychiatric disorders, providing a potential biological bridge between them (Palumbo et al., 2020; Wright et al., 2005).

Beyond systemic neuroendocrine modulation, stress may directly influence allergic inflammation at the tissue level. Mast cells, which are central effector cells in AR, express receptors for corticotropin-releasing hormone (CRH), a key mediator of the stress response (Cao et al., 2005). CRH released during stress can directly activate mast cells and enhance their degranulation. Moreover, mast cell activity may be modulated by additional stress-associated neuropeptides, including substance P and neurotensin (Alysandratos et al., 2012;

Toyoshima & Okayama, 2022). These interactions provide a mechanism through which psychological stress can locally amplify allergic inflammation independently of classical antigen-driven pathways.

The relationship between the nervous and immune systems in allergic disease is bidirectional. Activated mast cells release mediators such as histamine, tryptase, cytokines, and leukotrienes, which act on receptors expressed by sensory afferent neurons. This interaction may enhance neurogenic inflammation through axon reflex mechanisms, leading to increased vascular permeability, nasal hypersecretion, and mucosal edema. Furthermore, FcεRI receptors for IgE antibodies have been identified on certain peripheral nerve fibers, suggesting that neurons themselves may directly respond to allergen–IgE interactions (Undem & Taylor-Clark, 2014). Such mechanisms raise the possibility that allergen exposure could activate neural pathways independently of mast cell degranulation, potentially contributing to heightened symptom perception.

Chronic stress is also a well-established risk factor for psychiatric disorders, including depression and anxiety (Bower & Kuhlman, 2023). Given that stress can both promote Th2 polarization and directly activate mast cells, while allergic inflammation itself may influence brain function through cytokine signaling and neuroimmune pathways, a reciprocal relationship emerges. Psychological distress may exacerbate allergic inflammation, and the burden of allergic disease may in turn contribute to emotional and cognitive symptoms (Demoly et al., 2020; Kong et al., 2021).

Psychosocial mechanisms may further contribute to this relationship. Individuals suffering from seasonal allergic rhinitis may limit outdoor activities during periods of high allergen exposure, potentially leading to reduced physical activity, social withdrawal, and increased psychological stress (Meltzer, 2001). Notably, meta-analyses and randomized controlled trials have identified outdoor and nature-based activities and exercise as protective factors for mental health (Coventry et al., 2021; Heissel et al., 2023). Additionally, impaired sleep, diminished academic performance in children and adolescents, and reduced productivity in the workplace among adults may contribute to chronic stress, decreased self-esteem, and mood disturbances (Gómez-Jorge & Díaz-Garrido, 2022; Jarosz et al., 2020; Komnos et al., 2019).

The pediatric population appears to be particularly vulnerable to the negative effects of allergic rhinitis on mental health and daily functioning, because children and adolescents may possess fewer psychological and behavioral coping mechanisms to manage the challenges associated with chronic disease (Compas et al., 2012).

Collectively, these mechanisms support the existence of a self-perpetuating cycle in which stress and allergic inflammation reinforce one another. This may partially explain the frequent co-occurrence of allergic rhinitis and psychiatric symptoms and underscores the importance of integrating psychological factors into the comprehensive management of allergic disease.

Sleep Disturbances in Allergic Rhinitis

The mechanisms through which AR disrupts sleep appear to be multifactorial. First, inflammatory mediators involved in allergic responses may directly influence sleep regulation. Histamine and proinflammatory cytokines released during allergic inflammation are known to affect sleep architecture and circadian regulation (Çakan & Öztürk, 2022). Experimental studies in both humans and animal models have demonstrated that cytokines such as IL-1, IL-4, IL-6, IL-13, and TNF-α can alter both rapid eye movement (REM) and non-REM (NREM) sleep, increase sleep fragmentation, and prolong sleep onset latency (Irwin, 2019). These mechanisms may explain why patients with AR often experience non-restorative sleep and daytime fatigue despite having a total sleep duration comparable to that of healthy individuals (Liu et al., 2020).

Second, nasal obstruction itself represents an important mechanical contributor to sleep disruption. Nasal congestion can impair airflow during sleep, leading to difficulty falling asleep, frequent nocturnal awakenings, and reduced overall sleep quality. Consistent with this mechanism, patients with chronic nasal obstruction have been shown to exhibit higher rates of snoring and obstructive sleep apnea (Chirakalwasan & Ruxrungtham, 2014; Craig et al., 2010). Furthermore, studies have demonstrated that treatment of nasal obstruction with intranasal corticosteroids or decongestants can improve subjective sleep quality and reduce sleep-related symptoms (Lunn & Craig, 2011).

Comorbid conditions frequently associated with AR may further contribute to sleep impairment. For instance, allergic conjunctivitis can cause significant ocular itching and irritation, which may interfere with sleep initiation and maintenance (J. Li et al., 2023).

Importantly, the relationship between sleep disturbances and allergic inflammation appears to be bidirectional. Sleep deprivation and poor sleep quality have been associated with increased levels of

proinflammatory cytokines, including IL-6 and TNF-α (Veler, 2023). Such inflammatory changes may potentially exacerbate allergic inflammation and symptom severity, thereby further impairing sleep.

Sleep disturbances are also closely linked to mental health outcomes. Adequate sleep plays a crucial role in neuroplasticity, memory consolidation, learning processes, and emotional regulation. Consequently, chronic sleep impairment has been strongly associated with a wide range of psychiatric conditions, including mood disorders, anxiety disorders, and psychotic disorders (Palagini et al., 2022). Importantly, symptoms of insomnia have been identified as predictors of suicidal behavior (Rumble et al., 2020). Conversely, a meta-analysis (Scott et al., 2021) has shown that improvement in sleep quality was associated with reductions in depressive symptoms, anxiety, stress, and psychotic symptoms.

Conclusions

The proposed links between allergic rhinitis (AR) and mental health disturbances suggest that clinicians should consider a more integrative approach to patient care. Traditionally, AR has been perceived primarily as a localized inflammatory disorder of the nasal mucosa. However, epidemiological studies and emerging data from psychoneuroimmunology suggest that its effects may extend beyond the respiratory system, influencing sleep, cognitive functioning, emotional well-being, and overall quality of life.

Nevertheless, limitations of the current evidence should be acknowledged as the majority of available studies are cross-sectional, which precludes establishing causal relationships (Conway et al., 2024). Moreover, substantial heterogeneity exists across studies in terms of diagnostic criteria and assessment tools for both psychiatric symptoms and AR (Safia et al., 2024). Additionally, comorbid conditions may act as confounding factors. Finally, much of the mechanistic evidence is derived from animal models or non-allergic inflammatory paradigms, which may not fully reflect the complexity of human allergic disease. Further longitudinal and interventional studies are required to clarify the causal pathways and to determine whether effective management of AR may improve mental health outcomes.

From a clinical perspective, these findings emphasize the importance of recognizing the broader systemic and psychosocial burden associated with AR. Healthcare providers should remain aware that patients presenting with persistent or severe allergic symptoms may also experience fatigue, impaired concentration, sleep disturbances, anxiety, or depressive symptoms. Screening for sleep problems and psychological distress may therefore be beneficial, particularly in individuals with poorly controlled disease.

Optimizing the management of AR may have potential benefits extending beyond symptom relief. Effective control of nasal inflammation, through pharmacotherapy, allergen avoidance strategies, and allergen immunotherapy may improve sleep quality, cognitive functioning, and daily productivity. In pediatric populations, early recognition and treatment may be particularly important given that childhood and adolescence represent critical periods in neurodevelopment, during which the brain undergoes substantial structural and functional maturation. Disruptions in sleep, cognition, and emotional regulation during this stage may therefore have disproportionately large consequences (Baker & McMakin, 2024; Young et al., 2019).

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