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REVIEW ARTICLE

Psychoneuroimmunology of Stress: Evaluating the Therapeutic Role of Yoga-Based Mind-Body Integration- A Comprehensive Review

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ABSTRACT
The study of the intricate interactions of immunological responses, neuroendocrine pathways, and psychological stress is known as psychoneuroimmunology (PNI). Yoga, rooted in ancient Indian philosophy, is increasingly recognized as a mind-body intervention with scientific evidence of its potential to alter these pathways. The therapeutic potential of yoga in reducing stress-induced psychoneuroimmunological dysregulation and boosting immune resilience is evaluated in this review. Databases such as PubMed, Scopus, Web of Science, and Google Scholar were used to conduct a thorough literature assessment from 1985 to 2025. Studies examining the impact of yoga on immunological parameters, inflammatory cytokines, and neuroendocrine markers, particularly in stress-related settings, were the main emphasis of the inclusion criteria. Yoga has been shown to improve parasympathetic activity, lower cortisol and pro-inflammatory cytokine levels (such as TNF-α and IL-6), and downregulate the HPA axis. Additionally, yoga enhances mood-regulating neurochemicals and neuroprotective substances, promoting immunological and mental well-being. Incorporating yoga into medical care could offer a promising complementary strategy for immune regulation and stress reduction in patients with RA. To clarify the exact causes and improve protocols, further clinical research is necessary.
KEYWORDS: *Psychoneuroimmunology, Stress, Yoga, Mind–Body integration, Neuroendocrine pathways, Immune regulation, Inflammatory cytokines.*

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INTRODUCTION

Yoga and psychoneuroimmunology (PNI) represent converging paradigms that illuminate the intricate interdependence between the mind, body, and immune health. Originating in ancient India around 5000 BCE, yoga is a time-honored spiritual and philosophical system that has evolved into a widely recognized mind–body therapeutic approach. It encompasses physical postures (asanas), breath regulation (pranayama), meditation (dhyana), and ethical disciplines, all of which support holistic health. Asana practice, grounded in the Yoga Sutras and defined as *Sthira Sukham Asanam* (a stable and comfortable posture), aims not only to enhance strength, flexibility, and balance, but also to prepare the practitioner for deeper meditative states [1,2,3]. Modern research substantiates that yoga's integrative methodology promotes psychological stability and immune resilience. Its relevance as an evidence-based complementary therapy is increasingly acknowledged, particularly for managing stress-related and psychosomatic disorders. PNI is an interdisciplinary field that explores the bidirectional interactions between the central nervous system (CNS), the endocrine system, and the immune system, especially under conditions of psychological stress. PNI reveals how cognitive and emotional states influence immune function through neuroendocrine pathways, helping to decode the complex mind–body relationship in health and disease [4,5]. Recent findings demonstrate that stress and trauma impact immune regulation through neuroendocrine pathways such as the sympathetic–adrenal–medullary (SAM) axis and the hypothalamic–pituitary–adrenal (HPA) axis [6]. Cytokines, neuropeptides, and hormones serve as critical mediators, facilitating bidirectional communication between the brain and

immune cells [7]. Chronic activation of the HPA axis and autonomic nervous system (ANS) under stress has been linked to immune dysregulation, including suppressed immune surveillance and elevated systemic inflammation [8,9]. Acute stress responses are evolutionarily adaptive, priming cardiovascular, neuroendocrine, and musculoskeletal systems to manage perceived threats—originally predators, but now often psychological or medical challenges [8]. At the neurobiological level, central stress signals activate two key systems: the sympathetic nervous system (SNS) and the HPA axis. These pathways are regulated by corticotropin-releasing hormone (CRH) and locus coeruleus–norepinephrine (LC-NA) neurons, triggering the release of catecholamines and glucocorticoids that modulate immune responses. Concurrently, inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6) can stimulate these neuroendocrine systems, establishing a feedback loop between psychological states and immune activity [10]. Stress exerts its immunomodulatory effects through both direct neural innervation and hormonal signaling. Descending sympathetic fibers innervate primary lymphoid organs like the bone marrow and thymus, and secondary organs like the spleen and lymph nodes. These fibers release catecholamines that bind to adrenergic receptors on immune cells, modulating their function. The distribution of these receptors-particularly β2-adrenergic receptors, which are abundant on natural killer (NK) cells and less prevalent on T cells—indicates differential stress sensitivity across immune subsets [11,12]. Furthermore, the endocrine mediators released by the HPA and SAM axes—such as cortisol, epinephrine, norepinephrine, and prolactin—bind to receptors on white blood cells, thereby regulating immune processes [13]. Under prolonged stress, these hormonal cues alter leukocyte distribution, impair cytokine production, and suppress innate immunity. Maladaptive coping behaviors such as poor nutrition, substance abuse, and sleep disruption exacerbate this immunological imbalance [14]. Maier and Watkins (1998) proposed that stress-induced immune changes mimic those initiated by infections and contribute to "sickness behavior," characterized by fatigue, anhedonia, and social withdrawal. This response reflects a reallocation of energy toward immune defense, but under chronic stress, it dysregulates biological priorities, compromising health over time [15]. The significance of this study lies in its attempt to address the growing imperative to understand how psychological stress impacts immune function through neuroendocrine pathways, as explored by PNI. Contemporary findings further elucidate how these pathways mediate immune dysfunction and psychosomatic outcomes. This review synthesizes classical yogic wisdom and contemporary psychoneuroimmunological research to critically explore yoga's therapeutic potential in modulating neuroimmune pathways. By integrating these domains, the review aims to present a comprehensive understanding that supports incorporating yoga into modern healthcare systems for effective stress management and immune regulation.

MATERIAL AND METHODS

Methods

The literature on the function of yoga-based mind-body therapies in modifying stress-affected psychoneuroimmunological pathways was analyzed in this thorough review using a methodical and exacting approach. The methodology sought to guarantee the high-quality synthesis of the most recent scientific evidence, inclusivity, and methodological rigor.

Database Search

Several high-impact databases, including PubMed, Scopus, Web of Science, and Google Scholar, were searched with a specific focus. To document the historical development and the most recent developments in PNI and yoga research, literature published between 1985 and 2025 was included. The broad indexing of multidisciplinary biological, psychological, and integrative health studies in these databases led to their selection.

Search Keywords

To accomplish both breadth and specificity, the search employed Boolean operators (AND/OR) in conjunction with carefully chosen major and secondary keywords. The following were important terms: "mind-body medicine," "yoga," "stress," "immune modulation," "psychoneuroimmunology," "cortisol," "HPA axis," "parasympathetic activation," and "neuroendocrine pathways." Using this approach made sure that pertinent research relating to yoga's effects on immune system and neuroendocrine modulation under stress was retrieved.

Inclusion Criteria: Peer-reviewed English-language research that examines the effects of yoga therapies (such as asanas, pranayama, and meditation) on immunological function parameters, inflammatory cytokines (such as IL-6, TNF-α), and stress-related neuroendocrine indicators (such as cortisol, catecholamines). Cohort studies, systematic reviews, and randomized controlled trials (RCTs) were all taken into account.

Exclusion Criteria: Non-peer-reviewed publications, conference abstracts without complete data, research that only addressed general wellness or had nothing to do with immune regulation, and studies that lacked empirical data (such as theoretical articles or editorials) were not included.

Data Extraction and Synthesis

The data were extracted using a pre-made template that captured important information such as the type of yoga intervention (e.g., asana-based, pranayama-focused, or meditation), study populations (e.g., people with depression, autoimmune disorders, or chronic stress), primary biomarkers reviewed (e.g., cortisol, IL-6, TNF-α, and BDNF), length of the study, and the reported outcomes pertaining to immune modulation and stress reduction. An integrated and fact-based understanding of yoga's capacity to alter psychoneuroimmunological reactions was developed by synthesizing the gathered data. This strategy ensured sure that yoga's evaluation as a therapeutic mind-body intervention for immunological dysregulation caused by stress was extensive and supported by scientific evidence.

DISCUSSION

Psychoneuroimmunology of Stress: The study of the relationships among behavior, immunological processes, and brain and endocrine function is known as PNI [16]. The psycho-social-behavioral elements, stress, brain function (i.e., mind/thoughts), and physiological elements (i.e., neuroendocrine-immune system interactions) are all described under the psychoneuroimmunology model of health. The hypothalamic-pituitary-adrenocortical axis and the sympathetic-adrenomedullary (SAM) axis, which comprises the sympathetic nervous system (SNS), are the primary neuro-endocrine-immune pathways associated with stress [16,17]. Both axes become dysregulated as a result of ongoing stress. The HPA axis releases endocrine-based glucocorticoids, mainly in the form of the hormone cortisol, when chronic stress or trauma occurs because the SAM axis causes the SNS to produce norepinephrine, thereby starting a "fight or flight" reaction. Although it suppresses the immune system and eventually leads to chronic immunological dysregulation, high cortisol activity can be harmful to one's health [17,18].

Immune System and Adaptive Responses

The immune system is an intricate network composed of lymphocytes, macrophages, and other cellular components that protect the body from infections by bacteria, viruses, and parasites [19]. The first line of defense, innate immunity, involves NK cells, neutrophils, and macrophages, which are quickly mobilized to infection sites. These cells facilitate the activation and recruitment of pathogen-specific lymphocytes such as T and B cells that target and eliminate infections [20]. The main lymphocyte subsets mediating adaptive immunity include T-helper cells ($CD4^+$), T-cytotoxic cells ($CD8^+$), and B cells. T-helper cells coordinate immune responses by releasing cytokines that regulate other immune cells. T-cytotoxic cells identify and lyse cancerous or virus-infected cells. Upon activation, B cells differentiate into plasma cells that produce antibodies to neutralize pathogens and enhance phagocytosis [11,21]. Adaptive immunity comprises cellular and humoral components. Cellular immunity, primarily mediated by Th1 cells, involves cytokines such as IL-2 and interferon gamma (IFN-γ) that activate cytotoxic T cells and NK cells, targeting intracellular pathogens like viruses. Humoral immunity, mediated by Th2 cells through cytokines IL-4 and IL-10, activates B cells and mast cells to fight extracellular pathogens [11,21,22]. Five classes of immunoglobulins carry out distinct functions: IgA protects mucosal surfaces, IgE mediates allergic reactions, IgM is the first antibody during infection, IgG provides neonatal immunity, and IgD's role remains unclear [23]. Adaptive immunity also establishes immunological memory, crucial for defense against future exposures [24]. The initiation of adaptive immunity involves antigen-presenting cells like dendritic cells and macrophages detecting antigen-MHC complexes and triggering lymphocyte activation and cytokine-mediated immune coordination. Beyond immune cell specialization, the body's immune responses are intricately regulated by neuroendocrine Spathways, especially during stress responses [21].

Neuroendocrine-Immune Interactions and the Impact of Psychological Stress on Immune Function

Immune cells express receptors for hormones, neurotransmitters, and neuropeptides such as corticosteroids, insulin, prolactin, growth hormone, sex steroids, and β-adrenergic agents, allowing neuroendocrine modulation of immunity [10,25]. For instance, B lymphocytes express more β-adrenergic receptors than T cells, and CD4+ helper T cells express more than CD8+ cytotoxic cells [25,26]. These receptor expressions are dynamic and can change depending on immune cell activation status [7]. Peak stress hormone and neurotransmitter levels, as well as other physiological changes such as elevated blood pressure and heart rate, can all be indicators of how severe a stressor is. They can also influence how long these changes last, both during and after a stressor stops. It is crucial to remember that people differ greatly in how they perceive, interpret, evaluate, and cope with stress [8]. The neuroendocrine-immune axis regulates immune responses through complex signaling that depends on receptor

availability, cellular activation, and co-signal presence [10]. The physiologic stress reaction is the sole way a stressor can impact the body or brain, and this is crucial to realize. Although there are several contributing factors, the sympathetic nervous system's release of norepinephrine and epinephrine, as well as the hormones corticotropin-releasing, adrenocorticotropin, and cortisol, which occur after the hypothalamic–pituitary–adrenal axis is activated, are the main mediators of stress effects (Fig.1) [27]. Psychological and physiological stressors significantly affect immune function and disease susceptibility, including cancer, autoimmune disorders, and infections [9]. The hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) are principal pathways by which the central nervous system modulates immunity under stress [8].The HPA axis releases glucocorticoids such as cortisol, which suppress immune activation to prevent excessive inflammation but may impair long-term host immune defense under chronic stress conditions [28]. Simultaneously, catecholamines like norepinephrine, released through SNS activation, influence cytokine production, cytotoxic activity, and immune cell trafficking [10]. Prolonged or acute stress disrupts this balance, leading to immune dysfunction and increased vulnerability to disease (Fig.1) [11]. Glucocorticoids and catecholamines, primarily adrenaline and norepinephrine, are secreted systemically as a result of the hypothalamic CRH being released under stress. These substances then affect immunological responses. One could consider an immunological challenge to be a stressor if it jeopardizes the stability of the internal milieu. Thereby, CRH production is stimulated by cell products from an activated immune system, primarily the cytokines tumor necrosis factor (TNF)-α, interleukin (IL)-1, and IL-6, which in turn activate the HPA axis and the SNS (Fig.1).The immune system communicates bidirectionally with the CNS, transmitting immune status to the brain and integrating peripheral immunity with central regulation [7]. This mechanism provides a biological basis for how mental states influence immunity and overall well-being.

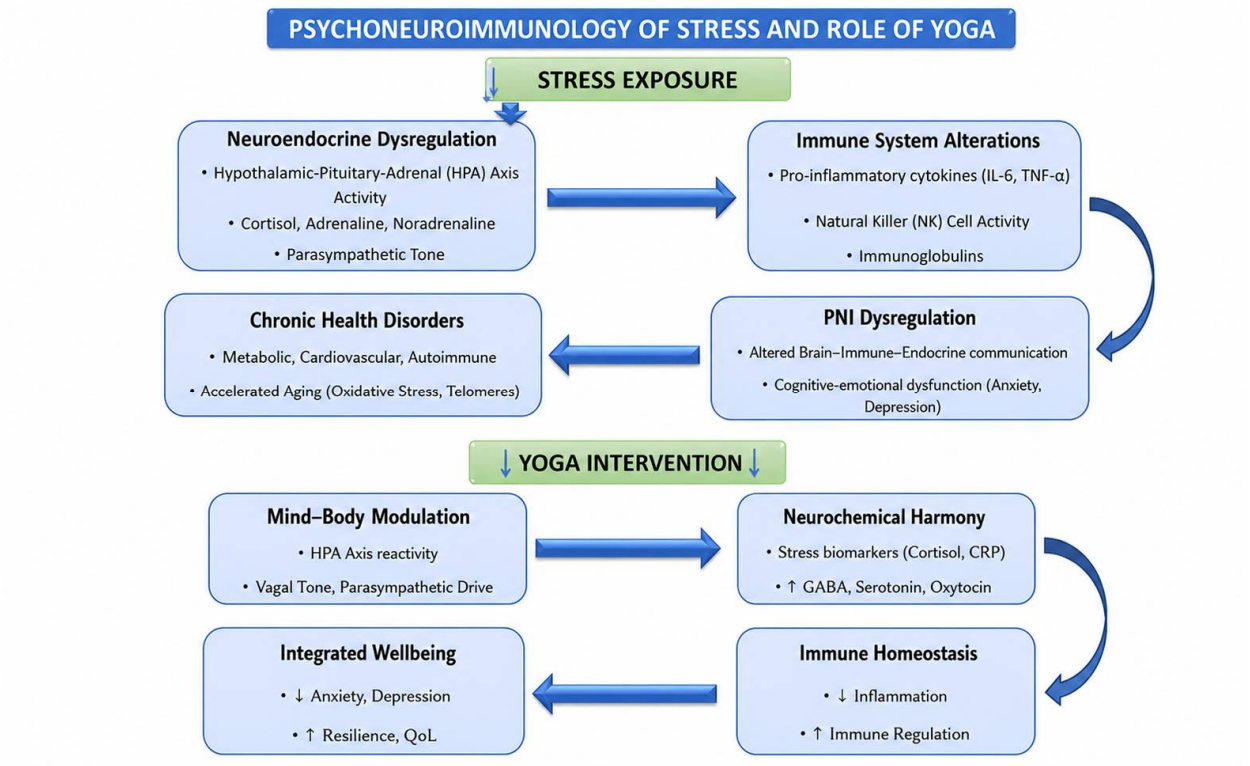

Fig. 1. Stress-induced psychoneuroimmunological disruption and yoga-based intervention for restorative regulation.

Psychoneuroimmunology: Integrating Mind-Brain, and Immunity

PNI studies the complex interactions between the nervous system, immune function, and psychological processes. This integrative field recognizes that immune activity can influence behavior and emotions, and conversely, mental states modulate immune responses [29]. Immunopsychiatry extends this concept by exploring immune system roles in psychiatric disorders. This reciprocal relationship emphasizes the mind-body connection in health, where emotional and psychological factors can modulate immunity and vice versa, providing a holistic framework for understanding stress-related diseases.

Yoga as a Comprehensive Mind–Body Modulator: Bridging Stress Reduction and Immune System Enhancement

Rooted in ancient Vedic and Upanishadic traditions, yoga is a mind-body practice aimed at reducing stress and promoting holistic health. Contemporary research shows yoga's potential in modulating immune function through neuroendocrine and psychological pathways. Recent research findings have demonstrated that yoga, as a holistic mind-body activity, can modify the immunological and neuroendocrine systems, mainly by lowering inflammation and stress (Fig.1). Combining asana (physical postures), pranayama (breathwork), and meditation is used to achieve this modulation, which affects immunological, neurological, and hormonal pathways [1,25,30]. This review highlights studies that concentrate on the physical practice of yoga and its impact on traditional immunological indicators, reflecting the disproportionate emphasis on the physical aspect of yoga (asana) in yoga research [30]. The field of PNI, or, going one step further, immunopsychiatry, acknowledges the immune system's role in regulating behavior and emotions. Current medical knowledge also acknowledges the impact of stress on the emergence of psychiatric disorders and interactions with the immune system [29].

Neuroendocrine Modulation: The hypothalamic–pituitary–adrenal (HPA) axis is the target of yoga, which also lowers levels of stress hormones like cortisol and adrenaline and increases mood-related neurochemicals like serotonin, oxytocin, GABA, and melatonin. Chronic stress-induced dysregulation of the HPA axis has been strongly linked to autoimmune and inflammatory diseases, such as rheumatoid arthritis, depression, and metabolic disorders. Yoga helps rebalance this dysregulation by producing a relaxation response (Fig.1). Tolahunase et al. [31] showed that an 8-week Yoga-Based Lifestyle Intervention (YBLI) increased BDNF and sirtuins, neuroprotective factors crucial for synaptic plasticity and cognitive resilience, while decreasing serum cortisol and DHEAS levels and reducing perceived stress in RA patients. Through yoga's impact on neuroendocrine signaling and gene expression profiles, these alterations reflect a transition from a sympathetic dominance prone to stress to a parasympathetic, restorative state [31,32].

Immune System Effects: Yoga practice has been shown to reduce inflammatory markers (e.g., IL-6, TNF-α) and improve immune parameters in individuals with chronic illnesses [33]. These effects may be mediated through reductions in stress hormones and improvements in autonomic nervous system balance. Recent clinical studies support yoga's role in enhancing immune responses and reversing stress-induced immunosuppression. For example, Zheng et al. [1,34] demonstrated that yoga interventions reduce stress-related immune suppression and improve overall immune function. These findings align with broader PNI research, which recognizes the interconnectedness of psychological stress, mental health, and immune function [29]. Yoga thus represents a promising integrative approach to managing stress and immune-related disorders.

CONCLUSION

Yoga's potential as an integrative mind-body treatment for modifying stress-related psychoneuroimmunological pathways is highlighted by this thorough review. Yoga provides a comprehensive approach to enhancing immunological and psychological well-being by lowering pro-inflammatory cytokines, improving neuroendocrine balance, and downregulating the HPA axis. Yoga may help manage stress-related diseases and boost immunity when included in traditional healthcare systems. To confirm these results and create uniform treatment procedures, more longitudinal and mechanistic studies are required.

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