



# Psychoneuroimmunological Interactions: The Impact of Psychological Stress on Immunological Function

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**Abstract:** Psychological stress plays a crucial role in modulating the immune system through complex mechanisms involving interactions between the nervous, endocrine, and immune systems. This review evaluates the impact of acute and chronic stress on immune function and the molecular pathways involved. This study employed a narrative literature review approach to explore the relationship between psychological stress and the immune system from a psychoneuroimmunological perspective. The literature review found that chronic stress has been shown to induce immune system dysregulation through activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, leading to increased levels of glucocorticoid and catecholamine hormones. These hormones influence immune cells, inhibiting the proinflammatory Th1 pathway, enhancing the Th2 response, and causing increased levels of inflammatory cytokines such as IL-6 and TNF- $\alpha$ . Consequently, stress contributes to an increased risk of chronic diseases such as cardiovascular disease, cancer, depression, and diabetes. Meanwhile, short-term acute stress can have adaptive immunostimulatory effects, especially in healthy individuals. The stress response is highly individualized and influenced by a person's biological vulnerability and coping capacity. By understanding the relationship between stress and the immune system in the context of psychoneuroimmunology, preventive approaches and clinical interventions can be developed to minimize the adverse effects of stress on physical and mental health.

**Keywords:** Immune system; inflammation; glucocorticoids; psychological stress; psychoneuroimmunology

## Introduction

Psychoneuroimmunology is an interdisciplinary field that examines the complex relationships between the central nervous, endocrine, and immune systems. This approach highlights the integrated, bidirectional communication pathways that link neurological, hormonal, and immunological responses to establish a balance between mind and body (Bower & Kuhlman, 2025; Hingorjo et al., 2025). One of the field's primary focuses is understanding how psychological stress affects health through modulating immune function.

The brain perceives psychological stress as a threat, activating the neuroendocrine system and triggering an immune response as part of the body's protective mechanisms (Mariotti, 2015). While acute stress can temporarily facilitate the innate immune response, chronic exposure has the opposite effect, causing immunological dysregulation, increasing proinflammatory cytokine levels, decreasing vaccination effectiveness, and slowing wound healing (Gouin, 2011; Tort, 2011). This imbalance triggers an increased risk of various chronic and inflammatory diseases, as well as exacerbating pre-existing immune

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and psychiatric disorders (Alotiby, 2024; Appleyard et al., 2020).

Through the activation of the hypothalamic-pituitary-adrenal (HPA) axis and increased release of glucocorticoids like cortisol, chronic stress is known to reduce the effectiveness of the immune system. Persistently elevated cortisol can suppress lymphocyte proliferation, inhibit antibody synthesis, and alter the balance of pro- and anti-inflammatory cytokines (Sandini et al., 2021; Stoffel et al., 2022). This immune imbalance explains an individual's susceptibility to opportunistic infections, autoimmune diseases, and progressive degenerative disorders.

In the context of modern life, work stress is a dominant factor contributing to chronic stress. Various studies have shown that individuals with high workloads, long working hours, and severe psychosocial pressure experience sleep disturbances, chronic fatigue, and even immune system disorders (Febriana et al., 2022; Firdaus et al., 2021). Epidemiological data indicate that more than two-thirds of the adult population experiences significant work stress annually, contributing to high rates of absenteeism, decreased productivity, and increased stress-related morbidity (Achmar et al., 2022; Tendean, 2020).

The COVID-19 pandemic has made matters worse by increasing workloads, especially in the healthcare industry, and exacerbating psychological distress because of social isolation, economic uncertainty, and infection risk (Hasibuan et al., 2021; Maradona & Syafwani, 2022). Significant increases in anxiety, despair, and burnout symptoms have allegedly been recorded among healthcare professionals directly involved in caring for COVID-19 patients, which has eventually affected their immunological function (Chen et al., 2022; Ichwanhaq, 2021). The prolonged stress response in this context demonstrates a close relationship between psychological stress and immune system changes, including increased levels of IL-6, TNF- $\alpha$ , and cortisol, contributing to endothelial dysfunction and chronic inflammation.

In addressing the widespread impact of stress on the immune system, psychological interventions such as talk therapy, stress management, meditation, and mindfulness approaches are important strategies for mitigating the immunosuppressive effects of stress (Tarigan et al., 2021). These approaches have lowered cortisol levels, dampened sympathetic nervous system activation, and restored immune function balance. The application of stress management also plays a significant role in supporting the care of patients with chronic diseases, such as cancer, which are heavily influenced by their psychological state (Marcelina & Yuliningtyas, 2021).

Based on the above description, it is important to review the existing literature on the impact of psychological stress on the immune system within a psychoneuroimmunological framework. This article aims to provide a comprehensive overview of the biological mechanisms underlying the relationship between stress and immunity and its clinical implications for physical and mental health. This understanding is expected to provide a scientific basis for developing more effective multidisciplinary prevention and intervention strategies to address the impact of stress on immunological function.

## Method

### *Literature study method*

This study used a narrative literature review approach to explore the relationship between psychological stress and the immune system from a psychoneuroimmunology perspective. A systematic literature search of scientific publications published between 2004 and 2024 focused on articles discussing the impact of acute and chronic stress on immune function, neuroendocrine mechanisms, and immune regulation through hormonal and cytokine pathways. Data sources were obtained from leading electronic databases, including PubMed, Scopus, and Google Scholar. Keywords used in the search process included "psychological stress," "immune system," "psychoneuroimmunology," "stress-induced inflammation," "glucocorticoid," and "Th1/Th2 shift."

### *Inclusion criteria*

Inclusion criteria included articles in Indonesian or English that were available in full-text form, were primary research articles, systematic reviews, or narratives, and relevantly addressed the interaction between stress and the immune system in humans or animal models. Articles lacking direct relevance, neither peer-reviewed nor available in a full version, were excluded from this review. The obtained articles were then selected and analyzed qualitatively based on thematic relevance and methodological strength. The chosen research findings were then grouped into key themes, such as activation of the hypothalamic-pituitary-adrenal (HPA) axis, the effects of glucocorticoids on immune cells, the shift from Th1 to Th2 responses, and the role of proinflammatory cytokines in mediating the relationship between stress and disease susceptibility.

Only articles from indexed and peer-reviewed journals were used as data sources to ensure the validity and credibility of the findings. The analysis was conducted using a critical and integrative approach to comprehensively understand the impact of stress on

immune system homeostasis and its potential clinical implications.

## Result and Discussion

### *Psychological stress as a trigger for psychosomatic disorders*

Psychological stress can manifest itself in physical illnesses, known as psychosomatic disorders, arising from mental factors or psychosocial events, particularly when individuals lack adequate coping skills (Sairah, 2021). Chronic stress alters the responses of the immunological and inflammatory systems, increasing systemic inflammation and suppressing immune function (Kinderlehrer, 2024). The hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system are both prolongedly activated in this process, which results in immunological dysregulation and heightened vulnerability to several infectious and chronic illnesses.

Chronic stress can exacerbate physical ailments and hasten the onset of chronic diseases, especially in people with pre-existing medical conditions (Schneiderman et al., 2005). The stressor's nature, length, and frequency significantly impact the association between psychosocial stress and health issues. Individual risk factors, including coping mechanisms, social support, and biological vulnerability, play a role. The body undergoes physiological wear and tear known as an allostatic load when stress is not followed by a period of relaxation or recuperation. This raises the chance of developing several diseases over time, including diabetes, cancer, cardiovascular disease, and mental health issues (Ghosh et al., 2022).

Chronic stress is also known to lower body image, limit mobility, and increase anxiety about potential health complications, all of which can create a recurring cycle of stress and disease (Kurdi et al., 2020). This persistent allostatic load triggers proinflammatory pathways and neuroendocrine changes directly impacting the immune system. Overactivation of the sympathetic nervous system leads to increased release of norepinephrine, which then influences immune cells through its interaction with adrenergic receptors, altering their activity, distribution, and function (Weber et al., 2022).

### *Immune system dysregulation due to HPA and sympathetic nervous system activation*

Studies have indicated that long-term stress raises proinflammatory cytokine levels, including TNF- $\alpha$  and IL-6, which are linked to the emergence of cardiovascular and metabolic disorders (Rohleder, 2014). This confirms systemic inflammation is a central pathway bridging psychological states and physical health status. In a prolonged state of alert, the body

experiences immune system disorders and significant physical and psychological problems (Montgomery & Gouvea, 2024). Therefore, stress can function both as a triggering factor and as an exacerbator of pre-existing pathological conditions (Yaribeygi et al., 2017).

It is important to understand that the stress response is highly individual and subjective, influenced by a person's coping capacity, health status, and biological vulnerabilities (Santos et al., 2020). Therefore, early detection of stress symptoms and development of interventions to mitigate its adverse impact are crucial in preventive medicine (Ping et al., 2024). The psychoneuroimmunology approach provides a robust scientific framework for understanding the interactions between the nervous, endocrine, and immune systems in stress. By understanding the molecular pathways involved, intervention strategies can be directed at modulating the stress response and strengthening the immune system, thus preventing more serious complications later in life.

### *Allostatic load and the risk of chronic disease*

Numerous studies have shown that coordinated, immunological, endocrine, and neurological systems react to psychological stress. This relationship is dynamic and adaptable in the short term, but may become problematic in long-term settings. The immune system operates in an unstable internal environment when the body's stress response system is repeatedly triggered. This environment is typified by cytokine imbalance, high cortisol levels, and altered innate and adaptive immune cell activity. This relationship's molecular pathways include HPA axis involvement, stress hormone release, and mediation by adrenergic receptors on immune cells.

### *Psychological impact and neuroendocrine changes on the immune system*

This phenomenon supports the hypothesis that chronic stress acts as a silent modulator of various pathological processes, including immunosenescence, insulin resistance, and even cancer progression. Prolonged elevations in IL-6 levels have been linked to inflammasome activation and the formation of an inflammatory microenvironment that supports abnormal cell growth. In addition, stress also decreases the immune response to new antigens, as shown by the reduced effectiveness of vaccination in individuals with high stress levels.

### *Proinflammatory cytokine pathways as mediators of immunological disorders*

These findings have broad clinical implications, particularly in holistic and preventive medicine. Treatment approaches that focus solely on the physical

aspects without considering the patient's psychological state can miss the root causes of many chronic diseases. Thus, building a multimodal treatment framework that integrates psychological interventions, including progressive relaxation, mindfulness, cognitive-behavioral therapy, and social support, is necessary. Such interventions have been shown to reduce inflammatory biomarkers and restore immune system homeostasis by restoring HPA axis function.

*The role of stress as a trigger and exacerbation factor for disease*

Future research in psychoneuroimmunology should focus more on identifying specific and predictive stress biomarkers for immunological changes and developing personalized interventions based on an individual's biological and psychosocial vulnerabilities. This review reinforces the urgency of a multidisciplinary approach to addressing stress as a key determinant of health and the importance of collaboration between immunologists, psychologists, and preventive medicine. Acute stress, in young, healthy individuals, is often adaptive and can trigger beneficial physiological responses, such as increased alertness, energy mobilization, and activation of the innate immune system. These responses allow individuals to cope with dangerous situations quickly and efficiently. However, when acute stress becomes excessive or is not followed by an adequate recovery phase, it can negatively impact health (Sharma et al., 2014).

*Individual variability in stress response*

Poorly managed acute stress can cause significant systemic dysfunction in individuals with poor health or older people. One consequence is desensitization of glucocorticoid receptors and disruption of the negative feedback mechanism of the hypothalamic-pituitary-adrenal (HPA) axis. This disruption leads to persistently elevated levels of stress hormones such as glucocorticoids and catecholamines, which impact the immune system and various other organs (Mariotti, 2015).

*The urgency of early detection and psychoneuroimmunological intervention*

Stress hormones are essential for regulating the immune system. Antigen presentation, immune cell proliferation, leukocyte migration, and the production of cytokines and antibodies are just a few of the components of immunity that glucocorticoids and catecholamines can impact. In acute stress, sympathetic nervous system activation and adrenal hormone release can suppress lymphocyte activity and proinflammatory cytokine production, particularly those associated with the Th1 response (Elenkov & Chrousos, 2006). At the

same time, the immune system switches from the proinflammatory Th1 response to the humoral and anti-inflammatory Th2 response.

*Dynamics of the interaction of the nervous, endocrine, and immune systems*

These changes reflect the body's adaptive mechanisms to limit tissue damage caused by excessive inflammation during periods of stress. However, when stressors persist chronically, these adaptive responses can become maladaptive and contribute to increased susceptibility to infection and other immune disorders (Alkadhi, 2013; Schneiderman et al., 2005). Furthermore, acute stress can impact behavior and psychological aspects, such as increased anxiety and sleep disturbances, which in turn indirectly impair immune function. The combined effects of neuroendocrine dysregulation and behavioral disturbances explain why acute stress, if not properly managed, can develop into a significant immunological and psychological burden.

*Chronic stress as a silent modulator and the risk of immunosenescence*

The short-term biological reaction to a temporary stressor is known as acute stress. Acute stress can be protective and temporarily improve immune function in healthy people, especially in young people with strong adaptive abilities. The sympathetic nervous system is activated during this reaction, and adrenal hormones like glucocorticoids and catecholamines are released. These hormones assist the body in mobilizing energy, boosting awareness, and fortifying its first line of defenses (Sharma et al., 2014). However, excessive and repeated stress—even in acute forms—can negatively impact immune function, particularly in older individuals or those with comorbid conditions. Prolonged activation of the stress system contributes to desensitization of glucocorticoid receptors and disruption of the negative feedback loop of the hypothalamic-pituitary-adrenal (HPA) axis, leading to the persistent secretion of stress mediators (Mariotti, 2015). This imbalance results in weakened immune control and tissue damage.

*Decreased Immune Response to New Antigens*

Stress hormones, particularly glucocorticoids and catecholamines, influence various aspects of the immune system, including inhibition of lymphocyte proliferation, regulation of leukocyte trafficking, decreased expression of MHC class II molecules, and modification of the cytokine profile (Elenkov & Chrousos, 2006). In general, acute stress suppresses the proinflammatory Th1-type immune response and promotes a shift toward Th2, which is more oriented toward humoral responses. This shift is an evolutionary

adaptation to protect the body from excessive inflammatory damage in emergencies, but can become dysfunctional if it persists or occurs in conditions of compromised health (Alkadhi, 2013; Schneiderman et al., 2005).

#### *Clinical Implications in Holistic and Preventive Medicine*

These findings confirm that acute stress is not immunologically neutral. Despite being traditionally seen as a defense mechanism, the acute stress response has wide-ranging and intricate effects on the immune system. Acute stress-induced activation of the sympathetic nervous system and the release of adrenal hormones can affect the immune system in two ways: first, they can boost innate immune activity (such as the activation of neutrophils and macrophages), but if the stressor is not promptly removed, adaptive immunity will be suppressed. One of the main impacts of repeated acute stress is an immunological trade-off, where the body sacrifices adaptive immune responses (including antibody production and specific T cell activation) in favor of a rapid stress response. This can reduce the body's ability to recognize new antigens, increase the risk of infection, and decrease the effectiveness of immunization.

#### *Immunological Shift from Th1 to Th2*

Recent studies also demonstrate that acute stress can change the immune cells' gene expression profile, causing proinflammatory genes to be expressed more and anti-inflammatory genes to be decreased. This is linked to metabolic disorders and an increased risk of chronic inflammation if stressors are repeated (Elenkov & Chrousos, 2006; Mariotti, 2015). The immunological shift from Th1 to Th2 dominance during acute stress also explains why individuals under severe stress are more susceptible to viral infections or the reactivation of latent viruses (such as herpes simplex or Epstein-Barr virus).

#### *Molecular Consequences: Immunological Trade-Off*

In the clinical context, it is important to distinguish the impact of acute and chronic stress in patient management, particularly those undergoing immunosuppressive therapy or vaccinations or facing high-stress situations (such as pre-operative patients, emergency department healthcare workers, or caregivers of chronically ill patients). This knowledge underscores the need for patient stress screening as part of a comprehensive immunological evaluation.

#### *Directions for Clinical Intervention and Future Research*

Psychologically based early intervention strategies, such as stress management training, breathing techniques, and cognitive-behavioral interventions, are also needed to help manage stress responses and

maintain immunological stability. The study of psychoneuroimmunology provides a critical theoretical and practical foundation for integrating biomedical and psychosocial approaches into comprehensive patient care.

## Conclusion

Both acute and persistent psychological stress can have a significant effect on the immune system. Chronic stress causes the long-term release of stress hormones such as catecholamines and glucocorticoids, which can impair immune function, increase inflammation, and cause several illnesses, including cancer, cardiovascular disease, and metabolic disorders. Conversely, acute stress in healthy individuals can temporarily support the immune response. However, if it occurs repeatedly or in vulnerable individuals, the effects can be detrimental and reduce the effectiveness of the adaptive immune system. The immune response to stress is also strongly influenced by biological conditions, the ability to manage stress, and social support. Therefore, early stress management through psychological and biomedical approaches is crucial to maintaining the body's balance and preventing long-term negative impacts. Maintaining a balanced nervous, hormonal, and immune system is key to maintaining health and preventing stress-induced diseases.

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The author declares no conflict of interest related to this article's research, writing, or publication.

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