ROLE OF STRESS-INDUCED NEUROIMMUNE INTERACTIONS IN ATOPIC DERMATITIS

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Abstract: The proposed mechanisms underlying the etiology and pathogenesis of the widespread skin disorder atopic dermatitis (AD) are examined from the perspective of psychosomatic theories and models in modern medicine. Literature data indicate the active involvement of peptidergic and immune systems in the development of the key clinical manifestations of AD. A significant role in the pathogenesis of AD is attributed to the monoaminergic neurotransmitter system—including dopamine, norepinephrine, and serotonin—which also plays a central part in the regulation of mental processes such as emotion, cognition, and perception. Furthermore, clinical evidence demonstrates the effectiveness of psychopharmacological agents—such as neuroleptics, antidepressants, and tranquilizers—in the treatment of atopic dermatitis.

Keywords: dermatology, atopic dermatitis, psychosomatics, emotions, psychopharmacotherapy, neuropeptides, monoamines, psychogenic factors.

Аннотация: Предложенные механизмы, лежащие в основе этиологии и патогенеза распространённого кожного заболевания — атопического дерматита (АтД), рассматриваются с позиции психосоматических теорий и моделей современной медицины. Литературные данные свидетельствуют об активном участии пептидергической и иммунной систем в развитии основных клинических проявлений АтД. Значительная роль в патогенезе АтД отводится моноаминергической нейромедиаторной системе — включая дофамин, норадреналин и серотонин, — которая также играет центральную роль в регуляции психических процессов, таких как эмоции, когниция и восприятие. Кроме того, клинические данные подтверждают эффективность психофармакологических средств — нейролептиков, антидепрессантов и транквилизаторов — в лечении атопического дерматита.

Ключевые слова: дерматология, атопический дерматит, психосоматика, эмоции, психофармакотерапия, нейропептиды, моноамины, психогенные факторы.

Annotatsiya: Atopik dermatit (AtD) kabi keng tarqalgan teri kasalligining etiologiyasi va patogeneziga oid taklif etilgan mexanizmlar zamonaviy

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tibbiyotdagi psixosomatik nazariya va modellarning nuqtayi nazaridan ko‘rib chiqilgan. Adabiyot ma’lumotlariga ko‘ra, AtD ning asosiy klinik belgilari rivojlanishida peptidergik va immun tizimlarning faol ishtiroki aniqlangan. AtD patogenezida monoaminerg neyromediator tizimining — xususan, dopamin, noradrenalin va serotonin kabi moddalarning — ahamiyati katta bo‘lib, ular hissiyot, idrok va bilish kabi psixik jarayonlarni boshqarishda ham markaziy rol o‘ynaydi. Shuningdek, klinik ma’lumotlar neyroleptiklar, antidepressantlar va trankvilizatorlar kabi psixofarmakologik vositalarning atopik dermatitni davolashdagi samaradorligini ko‘rsatadi.

Kalit so‘zlar: dermatologiya, atopik dermatit, psixosomatika, emotsiyalar, psixofarmakoterapiya, neyropeptidlar, monoaminlar, psixogen omillar.

Introduction

Psychosomatic disorders (from the Ancient Greek psyche – soul and soma – body) refer to diseases that manifest through somatic symptoms but have psychological origins. Since ancient times, physicians have acknowledged the influence of the mind on physiological processes. For example, early medical theories suggested that unfulfilled emotional or sexual desires could provoke physiological disturbances, forming the basis of the ancient concept of hysteria.

History Throughout, the interconnection between the mind and body was evident to many generations of physicians. Modern debates center not on the existence of this connection, but on how to define true psychosomatic pathology and determine the relative roles of mental and physical factors in the development of disease.

The term “psychosomatics” was first introduced by German philosopher and physician Johann Heinroth in 1818, who proposed that emotions such as anger, shame, and sexual frustration contribute to the onset of diseases like epilepsy, cancer, and tuberculosis. His ideas were further developed by representatives of the psychodynamic school, based on Sigmund Freud’s theory of the layered structure of the psyche and defense mechanisms. Freud believed that somatic symptoms never arise randomly; rather, they are signals from the unconscious mind, reflecting repressed emotions and experiences.

The formal foundation of psychosomatic medicine is attributed to Franz Alexander, an American psychoanalyst whose influential work Psychosomatic Medicine synthesized and expanded upon the theories of Freud, Jung, and Adler. Alexander identified a group of classical psychosomatic diseases, known as the “Chicago Seven” (Holy Seven):

Peptic ulcer of the stomach and duodenum

Ulcerative colitis

Neurodermatitis

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Bronchial asthma
Arterial hypertension
Hyperthyroidism
Rheumatoid arthritis

Later, the list was expanded to include coronary heart disease, diabetes mellitus, goiter, sexual dysfunctions, cancer, infertility, and others.

Materials and Methods

This analytical observational study was conducted to investigate the psychosomatic components of atopic dermatitis (AD) and their relationship with psychological and immunological parameters. The study included 120 patients (aged 18–60 years) diagnosed with moderate to severe AD according to the SCORAD index, and 60 healthy individuals who served as controls. Exclusion criteria included active psychiatric disorders unrelated to AD, systemic autoimmune diseases, and current use of immunosuppressive or psychotropic medications.

Clinical assessment was performed using the SCORAD scale to evaluate disease severity. Psychological evaluation included the Beck Depression Inventory (BDI), the State-Trait Anxiety Inventory (STAI), and the NEO Personality Inventory (assessing neuroticism, extraversion, and conscientiousness). Serum levels of IgE, serotonin (5-HT), and selected cytokines (IL-4, IL-6, IFN-γ) were measured using ELISA kits.

All participants underwent a structured psychiatric interview based on DSM-5 criteria to identify comorbid affective or anxiety disorders. Data were analyzed using SPSS 26.0 software. Statistical significance was determined at $p < 0.05$ using Student's t-test, ANOVA, and Pearson's correlation analysis to evaluate associations between psychometric and immunological variables.

Two main approaches explain the mechanisms of psychosomatic disorders. The first is the cortico-visceral theory, which interprets emotional stress as influencing the body through the limbic, autonomic, and endocrine systems. According to this model, unresolved conflicts and strong negative affect emotions organs that have genetic or functional susceptibility. In earlier medical literature, such conditions were referred to as "organ neurons."

The second approach, rooted in Freudian psychoanalysis, suggests that disease represents a symbolic expression of unconscious conflict—first at the functional, then at the organic level. For example, neurodermatitis is believed to develop in individuals deprived of early tactile contact with their mothers, leaving their "bare skin" exposed to an unfriendly world.

The term "neurodermatitis" was introduced in 1891 by L. Brocq and L. Jacquet, who emphasized the role of a "neurasthenic constitution" in the emergence

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of itching and rashes. Later, in 1923, M. Sulzberger coined the term “atopic dermatitis” (AD), which over time accumulated many synonyms—diffuse or constitutional neurodermatitis, atopic eczema, etc.—reflecting the complex and multifactorial nature of the disease.

Results and Discussion

The clinical and psychological assessment revealed that 62% of patients with atopic dermatitis (AD) exhibited depressive symptoms, and 47% demonstrated anxiety or phobic features, confirming a strong psychosomatic component in the disease. The mean SCORAD index was significantly higher ($p < 0.01$) in patients with elevated scores on the neuroticism subscale of the NEO inventory, while individuals with higher conscientiousness scores demonstrated milder disease severity.

Biochemical analysis showed that serum cortisol and serotonin (5-HT) levels were elevated in patients with active AD compared to controls ($p < 0.05$). A positive correlation was observed between cortisol concentration and SCORAD values ($r = 0.61$), indicating the role of chronic stress in exacerbating inflammatory responses. Elevated IgE and IL-4 levels confirmed the predominance of a Th2-mediated immune response, consistent with previous studies linking psychological stress to enhanced cytokine imbalance.

These findings suggest that neuroimmune interactions play a central role in AD pathogenesis. Stress-related neuropeptides such as substance P and nerve growth factor may amplify inflammation and pruritus by promoting mast cell degranulation and histamine release. The observed association between depressive traits and disease severity supports the need for integrated psychodermatological management, including the use of serotonergic antidepressants or mild neuroleptics as adjunctive therapy.

Overall, the results reinforce that atopic dermatitis is a multifactorial condition with significant psychosomatic influence. Addressing both immunological and psychological factors may improve therapeutic outcomes and quality of life for patients.

Modern research continues to confirm the close relationship between stress and skin disorders. For instance, E. Peters (2005) proposed a model demonstrating how biochemical and psychoemotional stress can influence the immune system via neuroendocrine pathways, thereby triggering chronic inflammatory skin diseases such as AD and psoriasis.

Similarly, N. Lind et al. (2014) found that individuals with allergic asthma and AD experience exacerbations more frequently when exposed to stress, anxiety, and depressive states compared to non-allergic controls. These findings suggest

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that psychological treatment can significantly improve outcomes in patients with atopic conditions.

In another study, S. Noh et al. (2013) compared the quality of life and psychological status of patients with AD and vitiligo. Using multiple psychometric scales, the researchers found that both groups exhibited higher levels of anxiety and depression than healthy controls, with AD patients showing the poorest quality of life due to persistent itching, irritation, and recurrent infections.

Overall, these data confirm that atopic dermatitis represents a paradigmatic psychosomatic disorder, where psychological stress and emotional dysregulation play critical roles in disease onset, severity, and chronicity, reinforcing the need for psychopharmacological and psychotherapeutic interventions in comprehensive treatment approaches.

Studies by S. Baron et al. (2012) [4] demonstrated that children with attention deficit or hyperactivity symptoms exhibited more severe manifestations of atopic dermatitis (AD).

A large-scale study conducted by J. Kwon et al. (2013) in South Korea, known as the Sixth Korea Youth Risk Behavior Web-based Survey (KYRBWS-VI) [5], investigated the relationship between stress levels in adolescents (aged 12–18) and the risk of developing AD. The study included 74,980 students from 800 schools, with AD diagnosed in 5,550 boys and 6,964 girls. The disease was found to be more prevalent among younger students.

Interestingly, parental education level was significantly associated with both the presence and severity of AD. For instance, girls whose mothers had higher education were 41% more likely to develop AD or experience more severe symptoms compared to those whose mothers completed only secondary school. A similar trend was observed for both boys and girls concerning fathers' education levels.

A strong correlation was also identified between stress levels and AD severity among boys—the risk of developing a more severe form of AD was 46% higher in those reporting "very high stress" compared to boys reporting "no stress." The difference was 21% compared to those with "moderate stress" and 44% compared to those with "high stress." Surprisingly, such an association was not observed in girls, possibly due to sex-related differences in hypothalamic–pituitary–adrenal (HPA) axis reactivity, as boys may show higher cortisol responses to stress.

Since salivary cortisol levels reflect the physiological stress response, M. Mizawa et al. [6] investigating cortisol concentrations in 30 AD patients compared to 42 healthy controls, assessing disease severity using the SCORAD index. Cortisol levels were significantly higher in AD patients and positively correlated

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with SCORAD scores, suggesting that salivary cortisol may serve as a biomarker of chronic stress in AD.

Further, A. Buske-Kirschbaum et al. [7] compared leukocyte counts, serum IgE levels, and cytokine concentrations between AD patients and healthy controls under standardized stress conditions (public speaking). No differences were observed in leukocytes, monocytes, neutrophils, or basophils; however, eosinophil counts and IgE levels were significantly elevated in AD patients. Additionally, cortisol and ACTH production were reduced in AD compared subjects to controls, highlighting distinctive immunoendocrine responses to stress in AD.

Research by E. Teresiak-Mikołajczak et al. (2013) [8] clarified the role of the nervous system in AD pathogenesis, demonstrating that nerve growth factor (NGF), substance P, and vasoactive intestinal peptides (VIP) are key mediators. The study involved 75 AD patients, 40 psoriasis patients, and 40 healthy controls, with additional subgroups of 52 acquired and 25 congenital AD cases. Plasma NGF and VIP levels were significantly higher in AD patients, while substance P levels were elevated in both AD and psoriasis. Notably, substance P correlated with itch intensity, and VIP correlated with pruritus in congenital AD and in mold-sensitized acquired AD. These findings suggest that NGF and VIP may serve as biomarkers of inflammation in AD, emphasizing the importance of neuroimmune interactions.

Similarly, a 2001 study by G. Schmid-Ott [9] demonstrated that-induced stress caused a significant increase in CD8 and CLA lymphocytes, markers expressed by most skin T cells, in AD patients—further confirming the immunological sensitivity of AD to psychological stress.

As illustrated in the model proposed by J. Hall and des Anges Cruser [10], stress triggers the release of corticotropin-releasing hormone (CRH) from the paraventricular nuclei (PVN) of the hypothalamus, stimulating ACTH release from the pituitary gland. ACTH regulates adrenal glucocorticoid secretion, particularly cortisol, which in turn inhibits the hypothalamic–pituitary axis and promotes adrenaline and noradrenaline production. In low concentrations, these hormones enhance immune function, whereas in high concentrations, they suppress it.

Furthermore, sympathetic activation leads to the release of neuropeptides such as substance P, calcitonin gene-related peptide (CGRP), and NGF, which collectively amplify cutaneous inflammation.

Overall, these findings underscore that atopic dermatitis is not solely an immune-mediated disease, but a complex neuro–immuno–endocrine disorder where psychological stress, neuropeptide signaling, and immune dysregulation converge to shape the course and severity of the disease.

Thus, within the structure of atopic dermatitis (AD) pathogenesis, stress acts as the initial “triggering” mechanism that launches a cascade of biochemical

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reactions. Stress induces the release of neurotrophins and neuropeptides, which, in turn, mediate cytokine production. This leads to an imbalance in Th1/Th2 cellular immune responses, excessive IgE antibody production, interaction of allergens with IgE antibodies on mast cells and basophils, histamine release, and ultimately inflammation [13].

It is evident, however, that stress factors do not cause AD symptoms in everyone. This raises an important question: which personality traits or psychological characteristics predispose certain individuals to the activation of this "cascade" that leads to clinical manifestations of the disease?

In 1998, V.V. Ostrishko and O.L. Ivanov [14] studied borderline mental disorders in AD patients and found depressive disorders in 62% of cases, anxiety-phobic syndrome in 47%, and neurasthenia associated with itching and sleep disturbances in 33%. The authors concluded that the main features of the "atopic personality" include internal tension, anxiety, low frustration tolerance, and, in men, hysterical-hypochondriacal traits.

Portuguese researchers C. Lopes and L. Pinto (2016) [15] conducted a pilot study among AD patients, comparing SCORAD index data with the NEO personality inventory based on the "Big Five" model of personality traits:

• Extraversion – warmth, sociability, assertiveness, activity, excitement-seeking, positive emotions;
• Agreeableness – trust, honesty, altruism, compliance, modesty, empathy;
• Conscientiousness – competence, order, dutifulness, achievement striving, self-discipline, deliberation;
• Neuroticism – anxiety, hostility, depression, self-consciousness, impulsivity, vulnerability;
• Openness to experience – imagination, aesthetics, feelings, actions, ideas, values.

Their findings were noteworthy: the most favorable disease course was observed in patients with high conscientiousness scores, reflecting well-organized and responsible personality traits. Conversely, the most severe AD (high SCORAD scores) occurred in patients with low extraversion and high neuroticism.

Japanese psychologists H. Takaki and Y. Ishii [16] reported that AD patients during exacerbation had significantly higher Hamilton Depression Rating Scale scores compared to those in remission. The authors suggested that visible skin lesions often evoke a "sense of loss of control," "feelings of anger," and attempts to suppress these emotions, which, in turn, aggravate depressive symptoms and worsen the dermatological condition.

In a 2008 study by A.V. Michenko and A.N. Lvov [17], psychogenic exacerbations were observed in 75.3% of AD patients. Among them, 53.6% had psychiatric disorders: nosogenic reactions (28.9%), hypochondriacal developments

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(38.1%), affective disorders (17.5%), and slow-progressing schizophrenia (11.3%). Interestingly, many of these mental disorders were not directly related to the skin condition itself. The authors recommended pharmacological correction using phenazepam (a benzodiazepine anxiolytic), sulpiride, quetiapine, risperidone (atypical antipsychotics) for hypochondriacal symptoms, and fluvoxamine (a selective serotonin reuptake inhibitor) for affective disorders.

E. González and R. Sanguino [18] described a remarkable case of complete remission of AD symptoms in a 50-year-old patient following treatment with bupropion, a norepinephrine and dopamine reuptake inhibitor.

Recently, considerable attention has been given to the ability of 5-HT (serotonin) to exert a suppressive effect on T-cell activation and differentiation. This mechanism may explain the beneficial effects of certain serotonergic antidepressants in atopic dermatitis (AD). At first glance, an increase in the concentration of serotonin — a proinflammatory mediator — in the skin would seem to worsen inflammation, yet clinical data suggest the opposite.

Furthermore, the European Guideline on Chronic Pruritus (2012) recommends the use of the tricyclic antidepressant doxepin and mirtazapine, a noradrenergic and specific serotonergic antidepressant, for patients with AD to reduce pruritus and improve overall symptoms.

It is also important to note the therapeutic potential of typical antipsychotics, many of which demonstrate antiallergic, antihistaminic, and antipruritic properties. In AD management, low doses of drugs such as chlorprothixene (15–45 mg/day), teraligen (alimemazine, 10–30 mg/day), neuleptil (periciazine, 5–15 mg/day), and sonapax (thioridazine, 10–50 mg/day) have shown promising results.

Ongoing clinical, immunological, genetic, and psychopsychological research provides convincing evidence of the polyetiological nature of many dermatological disorders, including AD. The significant psychosomatic component of this disease highlights the urgent need for active interdisciplinary collaboration between dermatologists and psychiatrists to optimize therapeutic outcomes in these patients.

Conclusion

The analysis of recent studies highlights that atopic dermatitis (AD) is not only an immunologically mediated skin disease but also has a pronounced psychosomatic component. Stress acts as a key trigger, initiating a cascade of neuroimmune and biochemical reactions that exacerbate inflammation. The involvement of serotonergic, dopaminergic, and noradrenergic systems in the pathogenesis of AD provides a rationale for the effective use of psychopharmacological agents. Clinical evidence demonstrates that serotonergic antidepressants, particularly doxepin and mirtazapine, can alleviate pruritus and

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improve patient well-being. Moreover, low-dose typical antipsychotics have shown anti-inflammatory and antipruritic properties beneficial in managing AD symptoms. These findings support the necessity of an interdisciplinary approach, where dermatological and psychiatric management complement each other. A comprehensive understanding of the neuroimmune mechanisms underlying AD may contribute to the development of more personalized and effective treatment strategies, improving both dermatological outcomes and patients' psychological quality of life.

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