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The Relationship Between Depression, Autoimmune Diseases and The Role of Exercise - A Literature Review

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Abstract

Introduction and purpose: Epidemiological evidence suggests a strong bidirectional relationship between depression and autoimmune diseases. The purpose of this review is to examine the shared neuroimmunological mechanisms linking these conditions and to evaluate the therapeutic role of physical exercise along with its clinical implications.

Materials and methods: Medical databases such as PubMed, Google Scholar were searched, data analyzed and put into the single article.

Description of knowledge: The gut-brain axis, vagus nerve signaling, and intestinal microbiota mediate the complex interactions between psychological and immunological processes. Dysregulation of the HPA axis, elevated pro-inflammatory cytokines (IL-6, TNF-α), and intestinal barrier dysfunction underlie both depression and autoimmune diseases like rheumatoid arthritis or inflammatory bowel disease. Physical exercise acts as a crucial modulator of these pathways, improving mood, functional capacity and lowering disease activity.

Conclusion: Recognition of shared neuroimmunological pathways highlights the need for integrated therapeutic strategies. Physical exercise should be implemented as a key non-pharmacological treatment that reduces depressive and autoimmune symptoms. Combining targeted physical activity with psychobiotics, anti-inflammatory diets along with pharmacological treatment, provides a comprehensive, patient-centered approach necessary for improving clinical outcomes.

Key words: IBD, depression, exercise, autoimmune diseases

1. Introduction and purpose

Nowadays, depression is a leading cause of the global burden of diseases, affecting approximately 5,7% of the global adult population, which corresponds to nearly 332 million people worldwide [1]. Epidemiological data indicate that depression frequently co-occurs with autoimmune diseases. Importantly, this relationship is bidirectional: the presence of an autoimmune disease is associated with a significantly higher risk of depression, while a history of affective disorders is linked to the development of autoimmune diseases later in life [2]. The prevalence of depression within this patient group varies depending on the specific condition; however, the highest rates are observed among those with inflammatory bowel disease (IBD). In this population, depressive symptoms not only affect patients with active disease, but were reported by about 25% of patients in remission [3].

The fundamental link between these conditions is chronic inflammation and complex interactions within the gut-brain axis [4]. Depression is associated with an increased production of pro-inflammatory cytokines and the dysregulation of the hypothalamic-pituitary-adrenal

axis, both of which may compromise the integrity of the intestinal barrier and alter the immune response. These disruptions may contribute to a bidirectional relationship between depression and autoimmune diseases, in which psychological and immunological processes are associated with one another [2]. A deeper understanding of the shared pathophysiological mechanisms linking depression and autoimmune diseases holds considerable clinical relevance, as it may enhance the ability of clinicians to identify depressive symptoms in patients with autoimmune conditions. Similarly, it's beneficial to recognize potential autoimmune involvement in patients presenting with affective disorders, as it supports earlier intervention and a more holistic approach to patient care. In this context, physical exercise serves as a therapeutic tool, capable of reducing both depressive and autoimmune disease activity. Physical activity is considered a safe and effective strategy supporting pharmacological treatment.

2. Description of the state of knowledge

2.1. Neurobiological pathways of the gut-brain axis: microbiota, vagus nerve signaling and stress response.

Modern science has moved away from the traditional view of the human body as a set of independent organs. Instead, neurobiological and gastroenterological research now emphasizes that our organism functions as an integrated network of profound interactions. Imperative to this new perspective is the gut-brain axis (GBA). It's a bidirectional system of communication which connects nervous, hormonal, metabolic and immunological signals [4, 5]. Due to the existence of GBA, the neurons in the brain influence the function of intestines along with the activity of immunology cells, at the same time, the signals from the digestive system have significant impact on the mood, cognitive functions and mental health [5, 6].

The central element of this bidirectional interaction is the intestinal microbiota. Extensive research has demonstrated that the microbiota is a key factor in regulating the stress response, particularly when the organism's homeostasis is disturbed [7, 6]. Communication within this axis is among others facilitated by highly specialized cells known as enteroendocrine cells (EECs). These cells monitor the intestinal lumen and, in response, secrete gut hormones such as cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1), and peptide YY (PYY), which

further influence the central nervous system (CNS). Additional mechanisms participating in this communication include tryptophan metabolism and the production of microbial metabolites, such as short-chain fatty acids (SCFAs). Furthermore, intestinal bacteria influence the synthesis of neuroactive compounds, including GABA and serotonin (the latter produced by enterochromaffin cells), which interact with epithelial receptors to modulate signals sent to the CNS [8]. Current literature highlights that the disruption of this delicate balance, named dysbiosis, may be associated with anxiety and depressive disorders, although findings remain heterogeneous and do not yet define a consistent microbial signature of depression [4, 9].

Another key factor connecting the gut-brain axis is the vagus nerve, the primary component of the parasympathetic nervous system. It acts as a major sensory conduit, transmitting information regarding the microbiota-derived signals via afferent fibers to the nucleus tractus solitarius (NTS) in the brainstem [10]. Furthermore, the vagus nerve possesses unique immunomodulatory properties mediated by the cholinergic anti-inflammatory pathway. The activation of this pathway has been shown to inhibit the release of pro-inflammatory cytokines by tissue macrophages, thereby playing an important role in systemic inflammatory regulation [11]. Chronic stress and depression has been associated with reduced vagal tone, which may influence the cholinergic anti-inflammatory pathway. This dysregulation can contribute to increased systemic inflammation, a factor implicated in the pathophysiology of various inflammatory and possibly autoimmune conditions [10].

Chronic stress, along with affective disorders, leads to the activation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in elevated cortisol levels and may increase the permeability of the intestinal epithelium [4]. This phenomenon, colloquially termed 'leaky gut,' may allow bacterial fragments, such as lipopolysaccharides (LPS), to penetrate the bloodstream and potentially trigger a systemic immune response [12]. In the context of autoimmune diseases, this mechanism serves as a bridge through which psychiatric conditions may aggravate inflammatory processes and modulate autoimmune activity within the organism. Understanding the role of the vagus nerve as a mediator and the microbiome as a regulator of the immune response is essential, as it opens new therapeutic perspectives for patients suffering from autoimmune diseases, specifically through interventions directed at both psychological health and the human microbiome [10, 11, 12].

2.2. The inflammatory theory of depression.

Clinical and epidemiological evidence demonstrate a co-occurrence of depression with chronic inflammatory diseases, which has prompted extensive research into the shared biological foundations of these conditions [13]. Clinically, depression manifests through symptoms such as anhedonia, sleep and appetite disturbances, and a chronic lack of energy, all of which significantly impair an individual's daily functioning [8]. The presence of treatment resistant depression highlights the need to better understand the pathophysiology of the disease [14]. Recent studies suggest the existence of distinct disease subtypes, notably an 'inflammatory subtype' of depression, characterised by immune dysregulation [15].

The relationship between depression and immunological conditions is increasingly recognized as clinically significant. Patients with inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, exhibit a higher prevalence of depression and anxiety disorders compared to the general population [13]. Intriguingly, this increased risk for mood disturbances is observed not only following the diagnosis of IBD but may precede the diagnosis, suggesting the presence of shared biological mechanisms between these conditions [13]. An important role in this relationship is played by the gut-brain axis and the dysfunction of the intestinal barrier [16].

Pro-inflammatory cytokines are thought to represent the biological link between depression and systemic inflammation. In patients with depression, particularly during the first episode or when the condition remains drug-naïve, frequently elevated levels of inflammatory markers such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP) are observed [15, 17]. These cytokines function as immune signaling mediators and may influence the central nervous system function. This neuro-immune interaction can induce "sickness behavior," characterized by symptoms such as anhedonia and fatigue [17]. Furthermore, cytokines like IL-6 and TNF-α activate the enzyme indoleamine 2,3-dioxygenase (IDO), which shifts tryptophan metabolism toward the kynurenine pathway and may contribute to reduced serotonin availability [18]. This metabolic diversion leads to a deficiency in serotonin while increasing levels of neurotoxic metabolites, such as quinolinic acid (QUIN). Elevated QUIN levels may cause neurotoxicity by acting as an NMDA receptor agonist [18]. What's worth mentioning is the fact that elevated levels of pro-inflammatory cytokines,

including IL-6 and TNF-α, are a well-established feature of autoimmune diseases such as rheumatoid arthritis and inflammatory bowel disease, where they play a role in driving chronic inflammation and tissue damage [19, 20].

One of the fundamental biological responses to depression and stress is mediated by the hypothalamic-pituitary-adrenal (HPA) axis. Chronic stress leads to hyperactivation of this pathway and the sustained secretion of cortisol [5]. Although cortisol physiologically exerts an anti-inflammatory effect, chronic depression has been associated with glucocorticoid receptor resistance, potentially impairing this regulatory mechanism [5, 16].
Consequently, this mechanism may impair the ability to regulate inflammatory processes, which may lead to an increased secretion of pro-inflammatory cytokines. Persistent activation of the HPA axis and associated inflammation may compromise the integrity of the intestinal barrier and disrupt the gut microbiota, further exacerbating immunological dysregulation. This evidence supports a link between depression and autoimmune diseases through common neuroimmunological mechanisms involving gut-brain axis [4, 13].

2.3. Depression and autoimmunological diseases: interconnected systems and clinical implications.

There is a bidirectional relationship between depression and autoimmune diseases, which is supported by numerous epidemiological analyses. Research indicates that patients suffering from depression may have an increased risk toward certain autoimmune diseases later in life, whereas the presence of an autoimmune disease is associated with a higher frequency of depressive episodes [2]. This relationship may be explained by shared pathophysiological mechanisms, such as chronic inflammation and immune system dysregulation. Consequently, these findings highlight a need for a holistic therapeutic approach for patients suffering from these comorbid conditions [2].

A meta-analysis demonstrates a significant relationship between autoimmune thyroiditis (AIT) and mental health disorders. Specifically, patients with AIT exhibit a higher risk of depression and anxiety compared to the general population [21]. It is suggested that the shared pathomechanisms of these conditions are rooted in the activation of inflammatory processes and the influence of immunological factors on the central nervous system [18, 21]. Additionally, the co-occurrence of these conditions may be linked to the intensity of systemic

inflammation and neurobiological dysregulation, although further research is needed to draw clearer conclusions [22].

Another area of significant neuroimmunological interaction is rheumatoid arthritis (RA). In this population, the prevalence of depression ranges from 14% to 48% and is associated with multiple clinical outcomes, including disease activity, pain severity, quality of life, and mortality [23]. Similar to other autoimmune diseases, the relationship between depression and RA appears to be bidirectional, with evidence suggesting that individuals with depression may have an increased risk of developing RA. This association may be explained by shared inflammatory mechanisms, as pro-inflammatory cytokines are involved in the pathogenesis of both depression and rheumatoid arthritis [23]. Furthermore, depression has been associated with alterations in subjective components of disease activity assessment, such as tender joint count and patient global assessment, both included in the Disease Activity Score 28 (DAS28). This has important clinical implications, as depressive symptoms may amplify the perceived severity of RA symptoms independently of objective inflammatory markers [24].

Depression is recognized as an important factor influencing clinical course of inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis. It is estimated that depressive symptoms occur in approximately 25% of IBD patients, a prevalence significantly higher than that observed in the general population [3]. These outcomes may be explained by disturbed gut-brain axis; research suggests that chronic stress may impair the function of the intestinal barrier and the immune response, thereby sustaining and intensifying systemic inflammation [25]. These results may contribute to a bidirectional feedback loop, with psychological distress and systemic inflammation driving the progression of both conditions [26].

In light of the presented evidence, new therapeutic approaches that simultaneously address the nervous and immunological systems are increasingly being considered. Particular attention is now directed toward psychobiotics, selected strains of intestinal bacteria that have been shown to modulate the gut-brain axis. Wallace and Milev in the systematic review suggested that treatment with probiotics may improve depressive symptoms by increasing serotonin availability and/or decreasing levels of inflammatory numbers, though clinical effects on patients with depression have yet to be studied [8, 27, 28]. Additionally, an anti-inflammatory diet may support conventional pharmacological treatment by influencing the intestinal microbiome and potentially improving mental well-being while aiding in disease management

[29]. A holistic approach may therefore be beneficial, integrating standard pharmacological treatment with psychological support and targeted dietary interventions to improve outcomes in patients with autoimmune conditions [30].

2.4. The therapeutic role of exercise in depression and autoimmune disorders

A meta-analysis by Noetel including 218 randomised controlled trials and more than 14,000 participants with depression, found that physical activity can be an effective treatment for reducing depressive symptoms and should be regarded as a key therapeutic option alongside psychotherapy and pharmacotherapy. The strongest and best-supported effects were observed for walking or jogging, yoga, and resistance training, while dance showed a large effect but was based on a much smaller evidence base. Yoga was found to be more effective among older adults, while strength training among younger people. An important finding was that higher exercise intensity was associated with greater improvement in depressive symptoms. These exercise interventions produced effects broadly comparable to cognitive behavioural therapy and, in this analysis, greater than those observed for SSRI monotherapy, although the authors note that the study was not designed to fully evaluate all antidepressant trials. Additionally, the study demonstrated that exercise increases the effects of SSRIs. The benefits of exercise were consistent regardless of the patient's age, sex, or initial severity of depression suggesting that exercise may be useful for a wide range of patients. When combined with pharmacotherapy, physical activity led to even better outcomes than either approach used in isolation, pointing to its value as part of a comprehensive, multimodal treatment plan [31].

A meta-analysis including 637 participants examined the effects of exercise on inflammatory bowel disease (IBD) [32]. The authors provide evidence of small-to-moderate improvement of disease activity, as patients who exercised showed less severe symptoms compared with control groups. Moreover, after 3–6 months of exercise, patients reported less fatigue, more energy, and better tolerance of daily activities. Exercise was also associated with improved muscle mass and reduced adipose tissue. What is very important is the fact that patients suffering from IBD were able to exercise without experiencing the exacerbation of the symptoms. Not only exercise affects the physical body and cardiorespiratory fitness, but also improves anxiety, depression, which is crucial, as the relationship between depression and inflammatory bowel disease is suggested to be significant. However, despite improvements in mood and disease activity, exercise did not significantly affect laboratory inflammatory markers or disease-specific quality

of life. Overall, the study suggests that exercise is a safe and beneficial addition to the treatment of IBD.

Exercise is demonstrated to be beneficial also for patients suffering from other autoimmune diseases, such as rheumatic arthritis (RA). Li and Wang demonstrated that exercise training for periods equal or more than 12 weeks can decrease disease activity among patients with RA [33]. Another study determined that aerobic exercise combined with strength training results in better aerobic capacity, physical function and fatigue, with hand exercise for better hand function [34]. For maximum results, different exercises are supposed to be selected depending on the patient's symptoms. It's worth emphasizing that any exercise is better than none. These findings are valuable for patients suffering from rheumatic arthritis.

3. Conclusion

Evidence supports the claim that there is a relationship between depression and autoimmune diseases. Among patients suffering from autoimmune diseases, depression appears to occur more frequently compared to the general population [2]. Researchers have proposed that this relationship may be mediated by several overlapping mechanisms, including gut-brain axis dysregulation, HPA axis dysfunction, and alterations in the gut microbiome, alongside the potential role of pro-inflammatory cytokines and intestinal barrier disruption, all of which carry important clinical implications [4, 9, 10, 12, 17]. The present review predominantly focuses on the association of depression with rheumatoid arthritis, autoimmune thyroiditis, and inflammatory bowel disease; however, further research is needed to elucidate its connection with other autoimmune conditions. Knowledge about the association of depression with other diseases is important for psychiatrists, as they may detect such a disease much earlier and refer to a specialist. This holistic approach is important not only for patients with autoimmune diseases, but also others, as the association e.g. of depression and endometriosis has also been suggested [35].

Furthermore, the integration of physical activity emerges as a non-pharmacological pillar in managing these comorbid conditions. Current evidence demonstrates that exercise is not only an effective tool for reducing depressive symptoms, comparable to or enhancing standard therapies, but it is also safe and beneficial for lowering autoimmune disease activity. By mitigating universal challenges such as fatigue and functional decline, structured exercise

interventions directly address both the psychological and physical burdens of these disorders [32, 33, 34].

Recognition of the shared pathophysiological mechanisms underlying depression and autoimmune diseases is of considerable significance, as it encourages a shift towards integrated diagnostic and therapeutic strategies that address both the psychological and immunological dimensions of autoimmune diseases. Ultimately, a comprehensive, patient-centred approach incorporating physical activity alongside standard treatments remains necessary for improving outcomes in individuals affected by both depression and autoimmune conditions.

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