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VITAMIN D AND INFLAMMATORY DISEASES OF THE SMALL AND LARGE INTESTINE: FROM MECHANISTIC INSIGHTS TO CLINICAL PRACTICE (REVIEW)

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Inflammatory diseases of the small and large intestine (IBD) constitute a major medical and social challenge due to their high prevalence, chronic course, and substantial impact on patients' quality of life. Immune dysregulation, alterations in the microbiome, and genetic susceptibility play central roles in the pathogenesis of these disorders, interacting within a complex network of inflammatory pathways.

Vitamin D, traditionally recognized for its role in calcium–phosphorus homeostasis, has emerged over recent decades as a key immunomodulatory and anti-inflammatory factor. It influences the integrity of the epithelial barrier of the small and large intestine, modulates cytokine production, and regulates T-cell activity. This article summarizes current evidence on the involvement of vitamin D in IBD pathogenesis, the high prevalence of its deficiency in affected individuals, and clinical trial data demonstrating the potential benefits of correcting vitamin D status. Particular attention is given to individualized dosing strategies, interactions with immunosuppressive agents, and the prospects of incorporating vitamin D into combined therapeutic regimens. The presented findings underscore the need for further research aimed at optimizing management strategies for IBD patients in the context of their vitamin D levels.

With growing interest in the role of micronutrients in maintaining immune homeostasis, evaluating the effects of vitamin D on pathophysiological processes in IBD is crucial for advancing personalized therapeutic approaches. The synthesized evidence provides a scientific foundation for the development of updated clinical guidelines and for deeper investigation of the relationship between nutritional status and the course of chronic inflammatory processes in the small and large intestine.

Key words: *inflammatory diseases of the small and large intestine, vitamin D, immunomodulation, pathogenesis, therapy, clinical practice.*

Людмила Каньовська, Альона Антонів, Юлія Яринич, Надія Паліброда, Володимир Паліброда. Вітамін D та запальні захворювання тонкої й товстої кишки: від механістичних уявлень до клінічної практики (огляд)

Запальні захворювання тонкої та товстої кишки (ЗЗК) становлять актуальну медико-соціальну проблему у зв'язку з високою поширеністю, хронічним перебігом та значним впливом на якість життя пацієнтів. У патогенезі цих станів ключову роль відіграють імунні порушення, мікробіомні зсуви та генетична схильність, які взаємодіють у складній мережі запальних каскадів.

Вітамін D, відомий своєю класичною роллю у регуляції кальцієво-фосфорного обміну, протягом останніх десятиліть розглядається як важливий імуномодулюючий та протизапальний фактор, що впливає на функціонування епітеліального бар'єра тонкої та товстої кишки, продукцію цитокінів та активацію Т-клітин. У статті проаналізовано сучасні дані щодо участі вітаміну D у патогенезі ЗЗК, частоти його дефіциту при цих хворобах, а також розглянуто результати клінічних досліджень, які підтверджують потенційну користь корекції вітамінного статусу. Окрему увагу приділено питанням індивідуалізації дозування вітаміну D, його взаємодії з імуносупресивними препаратами та перспективам використання у складі комбінованих терапевтичних стратегій. Представлений матеріал підкреслює необхідність подальших досліджень для визначення оптимальних підходів до менеджменту пацієнтів із ЗЗК з урахуванням рівня вітаміну D.

Враховуючи зростаючий інтерес до ролі мікронутрієнтів у підтримці імунного гомеостазу, аналіз впливу вітаміну D на патофізіологічні процеси при ЗЗК має важливе значення для розробки персоналізованих терапевтичних стратегій. Узагальнення представлених даних створює наукове підґрунтя для формування нових клінічних рекомендацій та поглибленого вивчення зв'язку між нутритивним статусом і перебігом хронічних запальних процесів у тонкій та товстій кишці.

Ключові слова: *запальні захворювання тонкої та товстої кишки, вітамін D, імуномодуляція, патогенез, лікування, клінічна практика.*

The urgency of the problem. Inflammatory bowel diseases (IBD), encompassing Crohn's disease and ulcerative colitis, rank among the most prevalent chronic non-infectious disorders of the small and large intestine, with a steadily rising global incidence. Over recent decades, their prevalence has markedly increased not only in high-incidence regions such as Europe and North America but also in previously low-prevalence areas, including Asia, the Middle East, and Latin America [1–3]. This epidemiological shift underscores the worldwide nature of IBD and reflects the influence of lifestyle and environmental determinants such as urbanization, dietary westernization, alterations in the microbiota of the small and large intestine, and psychosocial stress [4–5]. The chronic, relapsing-remitting course of IBD contributes to significant clinical complications, including strictures, fistulas, perforations, bleeding, and an elevated risk of colorectal carcinoma. Consequently, IBD imposes a substantial public health and socioeconomic burden, demanding an interdisciplinary approach to management [6].

The pathogenesis of IBD involves a multifactorial interplay between genetic susceptibility, immune dysregulation, microbial imbalance, and environmental triggers [7–8]. Disruption of innate and adaptive immune responses, along with an altered cytokine profile, perpetuates chronic mucosal inflammation [9]. Recently, increasing attention has been devoted to nutritional and metabolic regulators capable of modulating immune and epithelial function [10]. Among these, vitamin D has emerged as a critical immunomodulatory molecule, exerting regulatory effects beyond calcium–phosphate metabolism [11, 12]. The presence of vitamin D receptors (VDR) on epithelial, immune, and mesenchymal cells supports its pleiotropic role in anti-inflammatory signaling, mucosal defense, and tissue repair within the small and large intestine [13, 14].

Accumulating clinical and experimental evidence indicates that vitamin D deficiency correlates with increased disease activity, relapse frequency, and extraintestinal manifestations related to the small and large intestine in IBD patients [9, 15–16]. Furthermore, vitamin D contributes to homeostasis of the small and large intestine by maintaining the integrity of the epithelial barrier of the small and large intestine, regulating cytokine production, influencing T-cell differentiation, and supporting microbiome diversity [3, 10, 12]. Despite these insights, the optimal therapeutic dosing, duration, and interactions with immunosuppressive treatments remain under investigation [17,18]. Therefore, exploring the relationship between vitamin D status and IBD pathophysiology may pave the way toward personalized therapeutic strategies aimed at immune restoration and sustained remission. The role of vitamin D in the pathogenesis and treatment of IBD thus represents a crucial and promising field of modern gastroenterology.

The purpose: to summarize current evidence on the role of vitamin D in the pathogenesis, clinical course, and treatment of inflammatory diseases of the small and large intestine, including Crohn's disease and ulcerative colitis, and to evaluate its potential as an immunomodulatory and adjunctive therapeutic factor in clinical practice.

Research materials and methods: to develop a scientifically grounded review article, a systematic search of scientific publications was conducted across leading databases, including PubMed, Scopus, Web of Science, the Cochrane Library, and Google Scholar. The search utilized the following key terms: "vitamin D," "inflammatory diseases of the small and large intestine," "Crohn's disease," "ulcerative colitis," and "immune modulation." The literature search covered the period from 2005 to the present, reflecting the most recent advances in both fundamental and clinical gastroenterology.

The inclusion criteria encompassed randomized controlled trials, review articles, meta-analyses, experimental and clinical studies, as well as long-term cohort investigations assessing the role of vitamin D in the pathogenesis, clinical course, and treatment of inflammatory diseases of the small and large intestine. Publications that were irrelevant to the topic, insufficiently accessible, or demonstrated inadequate methodological quality were excluded from the analysis.

The analysis integrated data on vitamin D deficiency in patients with inflammatory diseases of the small and large intestine, polymorphisms of the vitamin D receptor (VDR), and the effects of vitamin D supplementation on disease activity and outcomes in Crohn's disease and ulcerative colitis. The synthesized findings were used to evaluate immunological mechanisms, clinical effectiveness, and the potential role of vitamin D as an immunomodulatory component within comprehensive therapy for patients with these disorders. This methodology provides a holistic understanding of the role of vitamin D in the pathogenesis, clinical management, and prevention of complications associated with inflammatory diseases of the small and large intestine.

Research results and their discussion. The global increase in the incidence of inflammatory bowel diseases (IBD) and the well-established influence of vitamin D deficiency on disease course underscore the importance of this topic for both clinical practice and medical research [1–3]. Vitamin D is an essential micronutrient whose primary physiological role involves maintaining calcium homeostasis and promoting bone mineralization. Current evidence confirms that vitamin D deficiency is widespread worldwide, including among the Ukrainian population [33–35]. The main sources of vitamin D are cutaneous synthesis under ultraviolet radiation, dietary intake, and pharmacological supplementation. Absorption primarily occurs in the small intestine, with the participation of bile acids, followed by lymphatic transport through chylomicrons into systemic circulation, where vitamin D binds to the vitamin D–binding protein (DBP) [13–18].

In the liver, vitamin D undergoes hydroxylation to form 25-hydroxyvitamin D [25(OH)D], the principal circulating form and biomarker of vitamin D status. The second hydroxylation in the kidneys produces 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], or calcitriol, the biologically active metabolite responsible not only for calcium–phosphorus metabolism but also for modulating immune responses and cytokine production [8,9]. Numerous studies have shown that low serum 25(OH)D levels correlate with more severe Crohn's disease and ulcerative colitis, frequent relapses, and extraintestinal manifestations [11–12]. Meta-analyses indicate that vitamin D supplementation in doses ranging from 2,000 to 5,000 IU/day reduces relapse risk and supports clinical remission, although optimal regimens remain to be standardized [13]. Epidemiological observations also suggest that individuals living at higher latitudes exhibit increased IBD incidence, indirectly supporting the role of sunlight exposure and vitamin D status as risk-modifying factors [16,32].

Vitamin D exerts immunomodulatory effects through vitamin D receptors (VDR) expressed in macrophages, epithelial cells, and lymphocytes, influencing the synthesis of antimicrobial peptides such as cathelicidin and maintaining the integrity of the epithelial barrier of the small and large intestine [27–31]. Deficiency shifts the immune response toward a pro-inflammatory phenotype, impairs lymphocyte maturation, and reduces secretion of antimicrobial factors, all of which may exacerbate mucosal inflammation. Experimental studies confirm that vitamin D deficiency disrupts microbial homeostasis in the large intestine, reducing Lactobacillus populations and increasing Clostridium and Bacteroides species, indicating that deficiency promotes dysbiosis and inflammation [17,35]. Moreover, vitamin D exerts systemic protective effects by lowering the risk of osteopenia, osteoporosis, arthropathies, and dermatological manifestations that frequently accompany IBD [15–16]. Both the European and American Gastroenterological Associations recommend routine monitoring of serum 25(OH)D levels in IBD patients and maintaining concentrations above 30 ng/mL [14].

These findings confirm that vitamin D is not merely a component of mineral metabolism but a critical regulator of immune homeostasis in IBD. Its deficiency is associated with more aggressive inflammation, higher relapse rates, and increased complication risks. Nonetheless, it remains unclear whether

vitamin D deficiency is a cause or a consequence of inflammation and malabsorption – or both, forming a "vicious cycle" that perpetuates disease activity [9–10]. Further prospective studies with standardized 25(OH)D baseline assessment are required to clarify these causal relationships.

Existing clinical evidence reveals a dose-dependent effect of vitamin D on inflammatory activity, yet the distinction between preventive and therapeutic dosing remains uncertain. Intervention trials using doses from 1,000 to 10,000 IU/day have yielded heterogeneous results, complicating the development of standardized treatment protocols. Factors such as therapy duration, seasonality, body mass, VDR gene polymorphisms, and concomitant immunosuppressive therapy influence clinical response and must be considered when designing supplementation strategies [17–18]. Establishing unified dosing recommendations and implementing structured vitamin D monitoring protocols are therefore essential for consistent clinical application. Large-scale, multicenter randomized controlled trials remain a major priority in this area.

The relationship between vitamin D and the microbiota of the small and large intestine warrants particular emphasis. Deficiency of this micronutrient is associated with reduced microbial diversity, increased pathogenic species, and impaired mucosal barrier function. Vitamin D modulates homeostasis of the small and large intestine through the regulation of antimicrobial peptides, mucins, and cytokines - key components that shape microbial composition and immune tolerance [1–5, 17, 35]. Maintaining optimal vitamin D levels may therefore not only suppress inflammatory activity but also restore microbial balance, contributing to stable clinical remission. This interplay highlights vitamin D as a pivotal link between nutrition, immunity, and mucosal integrity, providing a strong rationale for its integration into comprehensive management strategies for inflammatory diseases of the small and large intestine.

Conclusions. Both epidemiological and experimental studies indicate that vitamin D plays a significant role in the pathogenesis of inflammatory diseases of the small and large intestine (IBD), as well as in colitis-associated colorectal cancer [2–4, 9–11]. Although vitamin D is known to regulate immune cell transport and differentiation, epithelial proliferation, barrier function, and antimicrobial peptide synthesis [3, 7, 10, 12, 13], the precise mechanisms by which it influences the course of colitis remain incompletely understood. Most existing evidence suggests protective associations between optimal vitamin D levels, IBD activity, and a reduced risk of colorectal neoplasia associated with inflammation of the large intestine [9, 10, 15, 16]. However, experimental studies evaluating vitamin D as a therapeutic agent have produced inconsistent results, likely due to variability in model systems and the multifaceted role of vitamin D in immune regulation and maintaining homeostasis in the small and large intestine [3, 9, 17]. Considering that vitamin D supplementation in patients with inflammatory diseases of the small and large intestine is simple, inexpensive, and well tolerated, further investigation is warranted to elucidate its mechanisms of action and identify patient populations most likely to benefit from such adjunctive therapy.

Future research should focus on establishing individualized dosing regimens that account for patient body weight, seasonality, concomitant medications, and genetic background. Another promising direction is the study of vitamin D interactions with biologic agents, including anti-TNF and anti-IL-12/23 therapies, which are integral components of current management of inflammatory diseases of the small and large intestine [14, 17, 18]. Furthermore, exploration of vitamin D receptor (VDR) gene polymorphisms may offer predictive markers for treatment response and guide personalized therapeutic approaches. Integrating serum 25(OH)D monitoring into clinical algorithms could enhance disease control, reduce relapse rates, and improve long-term outcomes.

In summary, vitamin D should be considered not merely a component of mineral metabolism but a key immunomodulatory and metabolic factor capable of influencing both local and systemic manifestations of inflammatory diseases of the small and large intestine. Nevertheless, confirmation of these findings requires large-scale, multicenter, randomized controlled trials that take into account genetic variability, environmental influences, and microbiome composition [15–18]. Continued exploration of these interrelations represents a promising frontier in modern gastroenterology and may ultimately establish vitamin D as a fundamental component of comprehensive therapy for inflammatory diseases of the small and large intestine.

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