ORIGINAL ARTICLE

INTERNATIONAL JOURNAL OF ARTIFICIAL INTELLIGENCE

ISSN: 2692-515-x, Impact Factor: 9.23

American Academic publishers, volume 6, issue 03,2026

Journal: https://www.academicpublishers.org/journals/index.php/ijai

APPLICATION OF VITAMIN D IN BRONCHIAL ASTHMA

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Abstract

Bronchial asthma is a common chronic inflammatory disease of the airways characterized by bronchial hyperreactivity. Data have been obtained on the role of vitamin D deficiency in the pathogenesis of this disease. A genomic association between vitamin D and asthma has been established. Vitamin D exerts anti-inflammatory and immunomodulatory effects. In pregnant women with insufficient intake of antioxidants and vitamins D and E, there is a high risk of giving birth to children with Atopic dermatitis and bronchial asthma. The administration of vitamin D to children helps reduce respiratory diseases and alleviate the course of asthma.

Keywords

vitamin D, bronchial asthma, pathogenesis, disease course, supplementation, bronchial obstruction

Bronchial asthma (BA) is the most common chronic inflammatory disease of the airways, characterized by periodically occurring bronchial obstruction that resolves completely or partially either spontaneously or under the influence of therapy. In childhood, BA accounts for more than 90% of all cases of bronchial obstruction syndrome. In addition to bronchial constriction, the obstructive syndrome in BA is caused by edema of the bronchial mucosa, hypersecretion, and dyscrinia (excessive production of viscous mucus). The obstructive syndrome is usually associated with increased airway reactivity in response to various external and endogenous stimuli in the context of atopy.

Over the past 5–10 years, concepts of asthma pathogenesis and therapy have significantly evolved. It has been established that, along with genes responsible for immunoglobulin E (IgE) production and bronchial reactivity, the status of certain vitamins and the receptor systems responsible for their utilization also play an important role.

The classical view of vitamin D as a compound necessary only for skeletal formation has been replaced by an understanding of its role not only in the normal functioning of various organs and systems, but also in the development of atopy and bronchial asthma.

The effect of vitamin D deficiency on the development of asthma begins in utero. Adequate intake of antioxidants and vitamins E and D during pregnancy reduces the likelihood of asthma development in the child.

There is also an inverse relationship between maternal vitamin D levels and the development of allergen-specific IgE antibodies in children during the first two years of life, as well as the formation of Atopic dermatitis and bronchial asthma within the following four years. Vitamin D supplementation leads to a decrease in the pro-inflammatory cytokine interleukin-17

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(IL-17). A genomic influence of vitamin D on asthma development has been demonstrated, with polymorphisms in the vitamin D hydroxylase gene strongly correlating with asthma in children.

Vitamin D participates in the formation of local immunity in the respiratory tract, actively influencing the biological functions of lung cells. This is supported by the high expression of CYP27B1, which is responsible for vitamin D activation, in pulmonary epithelial cells. In addition, polymorphisms of the vitamin D receptor correlate with susceptibility to respiratory infections. Vitamin D deficiency is considered a risk factor for asthma, comparable to factors such as urban living and obesity.

One of the most important functions of vitamin D is the suppression of T-helper type 9 cell activity, which produce pro-inflammatory cytokines such as IL-9, IL-5, and IL-13. Another mechanism involves increasing steroid sensitivity by enhancing IL-10 secretion from regulatory T cells, while simultaneously reducing the expression of the FKN gene associated with steroid resistance. Notably, the combination of vitamin D and dexamethasone is significantly more effective than dexamethasone alone. This effect is partly related to the ability of vitamin D to reduce hyperplasia of airway smooth muscle cells.

Hyperplasia and proliferation of smooth muscle cells are key factors in chronic bronchial obstruction and airway remodeling, mediated by vascular endothelial growth factor, fibrinogen, and IL-6. Vitamin D suppresses these stimulators, promoting DNA repair and preventing progression to the S-phase of the cell cycle. It also increases the synthesis of cathelicidin, a peptide of the innate immune system that inhibits viruses, fungi, gram-positive and gram-negative bacteria, and Mycobacterium tuberculosis, suggesting a protective role in infection-induced asthma.

In lung tissues, vitamin D exerts anti-inflammatory effects, significantly reducing eosinophil counts in sputum without affecting neutrophil levels. Studies have shown that in control groups receiving placebo, eosinophil levels increased.

Genes responsible for vitamin D metabolism are associated with genes involved in asthma pathogenesis, indicating a genomic link between vitamin D and the disease. Experimental studies in mice lacking the vitamin D receptor in lung tissue demonstrated increased collagen deposition and neutrophil accumulation, leading to lung tissue hyperplasia.

Disruption of vitamin D interaction with its receptor, particularly in deficiency states, leads to impaired homeostasis and dysfunction of alveolar macrophages, which can induce tissue proliferation and contribute to the development of emphysema.

In this context, vitamin D supplementation in patients with bronchial asthma appears justified, along with evaluation of its clinical effects. Studies conducted in countries such as the United States, Qatar, and Costa Rica confirm vitamin D deficiency in children with asthma. This deficiency is a predictor of asthma development, severity, and increased frequency of exacerbations. Observational data from 100 children with asthma showed that administration of 60,000 IU of vitamin D monthly contributed to reduced disease severity and improved asthma control.

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Asthma is a chronic respiratory disease characterized by airway inflammation and hyperreactivity, affecting millions of people worldwide.

Vitamin D is involved in the pathogenesis of bronchial asthma, exerting anti-inflammatory and immunomodulatory effects. Low levels of vitamin D (less than 20 ng/mL in serum) are associated with various respiratory diseases, including asthma.

Studies on vitamin D supplementation are ongoing; however, their results are not always consistent. Further research involving multidisciplinary teams of geneticists, clinicians, and nutritionists is required.

Conclusion:

Vitamin D plays an important role in the pathogenesis of bronchial asthma by exerting anti-inflammatory and immunomodulatory effects, reducing the activity of pro-inflammatory cytokines, and enhancing steroid therapy responsiveness. Vitamin D deficiency is associated with increased frequency of exacerbations, greater disease severity, and the development of bronchial obstruction syndrome. Supplementation of vitamin D in children and pregnant women demonstrates a positive effect on the prevention and control of the disease. However, study results are inconsistent, highlighting the need for further clinical and genetic research to determine the optimal doses and regimens of vitamin D in the comprehensive management of bronchial asthma.

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