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THE EFFECT OF VITAMIN D ON MORPHOFUNCTIONAL DEVELOPMENT AND THE IMMUNE SYSTEM IN CHILDREN

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Abstract

Vitamin D is a crucial fat-soluble micronutrient that plays a significant role in the regulation of bone metabolism, muscle function, and immune system activity, particularly in children. In recent years, increasing attention has been paid to the broader biological effects of vitamin D beyond skeletal health. This study aims to analyze the impact of vitamin D on morphofunctional development and immune function in pediatric populations[1]. Vitamin D deficiency remains a widespread global health issue affecting both developed and developing countries. Insufficient levels during childhood are associated with impaired growth, skeletal deformities, reduced bone mineralization, and increased susceptibility to infections. Furthermore, emerging evidence suggests that vitamin D is involved in immune modulation and may reduce the risk of autoimmune diseases and chronic inflammatory conditions. The metabolism of vitamin D involves its synthesis in the skin under ultraviolet B radiation, followed by activation in the liver and kidneys. Serum 25-hydroxyvitamin D is considered the main indicator of vitamin D status[2]. Epidemiological data indicate a high prevalence of deficiency among children and adolescents, particularly in regions with limited sun exposure or inadequate dietary intake[3].

Keywords

Vitamin D, children, morphofunctional development, immune system, deficiency, bone metabolism, pediatric health.

Introduction. Vitamin D is a fat-soluble micronutrient that plays a crucial role in regulating various physiological processes in the human body, including bone tissue formation, muscle function, and immune system activity. While its classical function has long been associated with calcium and phosphorus metabolism and skeletal development, recent scientific findings have demonstrated that vitamin D is also involved in the pathogenesis and prevention of numerous chronic diseases. Following the fortification of food products with vitamin D and the apparent decline in rickets, many healthcare professionals assumed that vitamin D deficiency had been effectively eliminated[4,5]. However, contemporary research indicates that vitamin D deficiency remains widespread among both children and adults. During fetal development and childhood, inadequate vitamin D levels may lead to growth retardation, skeletal deformities, and an increased risk of fractures later in life. In adults, vitamin D deficiency contributes to osteopenia, osteoporosis, osteomalacia, muscle weakness, and a higher likelihood of bone fractures[6]. The presence of vitamin D receptors in most tissues and cells, as well as the discovery of enzymatic systems capable of converting 25-hydroxyvitamin D into its biologically active form, 1,25-dihydroxyvitamin D, has significantly expanded the understanding of its biological functions[8]. These findings suggest that vitamin D plays an important role in reducing the risk of various

chronic conditions, including cancer, autoimmune disorders, infectious diseases, and cardiovascular diseases.

Materials and Methods. This study was conducted using a comprehensive analytical and descriptive approach based on the review of recent scientific literature and clinical data related to vitamin D in pediatric populations. Relevant articles were selected from international scientific databases, including PubMed, Scopus, and Web of Science, focusing on publications from the last decade. The inclusion criteria consisted of peer-reviewed studies addressing vitamin D metabolism, deficiency prevalence, and its effects on morphofunctional development and immune function in children. Both observational and experimental studies were considered. Key indicators analyzed included serum levels of 25-hydroxyvitamin D, bone mineral density, growth parameters, and immune response markers. Data were systematically analyzed and synthesized to identify common patterns, correlations, and clinical implications. Statistical data from epidemiological studies were also incorporated to assess the global prevalence of vitamin D deficiency among children and adolescents.

Sources and Metabolism of Vitamin D. Vitamin D is obtained through three primary sources: sunlight exposure, dietary intake, and nutritional supplements. Ultraviolet B (UVB) radiation with wavelengths between 290 and 315 nm penetrates the skin and converts 7-dehydrocholesterol into previtamin D_3, which is subsequently transformed into vitamin D_3. Excess vitamin D produced in the skin is naturally degraded by sunlight, preventing toxicity from sun exposure. Only a limited number of natural food sources contain significant amounts of vitamin D, with fatty fish being one of the richest sources. The term “vitamin D” refers to two major forms: vitamin D_2 (ergocalciferol), derived from ultraviolet irradiation of ergosterol in yeast, and vitamin D_3 (cholecalciferol), synthesized from 7-dehydrocholesterol obtained from lanolin. Both forms are commonly used in over-the-counter supplements, although vitamin D_2 is often prescribed in clinical settings[9]. Vitamin D obtained from the skin and diet undergoes metabolism in the liver to form 25-hydroxyvitamin D, which is the primary circulating form and the main indicator of vitamin D status. Subsequently, in the kidneys, it is converted into the biologically active form, 1,25-dihydroxyvitamin D, by the enzyme 1α-hydroxylase (CYP27B1). This process is tightly regulated by parathyroid hormone, as well as serum calcium and phosphorus levels. Fibroblast growth factor 23 (FGF-23), produced by bone tissue, reduces phosphate reabsorption and inhibits the synthesis of active vitamin D[10,11]. In turn, 1,25-dihydroxyvitamin D enhances intestinal absorption of calcium and phosphorus and promotes renal calcium reabsorption. It also induces the expression of 24-hydroxylase (CYP24), which converts active vitamin D metabolites into inactive forms[12].

Definition and Prevalence of Vitamin D Deficiency. Although there is no universal consensus on optimal serum levels of 25-hydroxyvitamin D, most experts define deficiency as levels below 20 ng/mL (50 nmol/L). Levels between 21 and 29 ng/mL are considered insufficient, while levels of 30 ng/mL or higher are regarded as sufficient. Vitamin D toxicity is typically observed at levels exceeding 150 ng/mL (374 nmol/L). Serum 25-hydroxyvitamin D levels are inversely correlated with parathyroid hormone (PTH) levels until concentrations reach approximately 30–40 ng/mL, after which PTH levels stabilize[13]. Increasing vitamin D levels has also been shown to significantly improve intestinal calcium absorption. Globally, approximately one billion people are estimated to have vitamin D deficiency or insufficiency. Studies indicate that 40–100% of elderly individuals in the United States and Europe have low vitamin D levels[14]. Among postmenopausal women receiving treatment for osteoporosis, more than 50% have

serum levels below the optimal threshold. Children and adolescents are also at high risk. For example, studies conducted in Boston revealed vitamin D deficiency in 52% of Hispanic and African American adolescents, while research in Maine showed deficiency in 48% of white adolescent girls[21]. Seasonal variation plays a significant role, with deficiency rates increasing during late winter[15]. In Europe, the limited fortification of food products contributes to higher deficiency rates. Even in regions with abundant sunlight, such as the Middle East and Asia, vitamin D deficiency remains prevalent due to reduced sun exposure. Studies in countries such as Saudi Arabia, the United Arab Emirates, India, and Turkey have reported deficiency rates of 30–50%. Pregnant and lactating women are also at increased risk. Despite supplementation, a large proportion of women and their newborns exhibit vitamin D deficiency at birth[23]. Furthermore, longitudinal studies have demonstrated that low baseline vitamin D levels are associated with an increased risk of certain cancers, including colorectal cancer[18].

Results. The analysis revealed that vitamin D deficiency is highly prevalent among children worldwide, with reported rates ranging from 30% to over 50% in different populations. The findings demonstrated a strong association between low serum 25-hydroxyvitamin D levels and impaired bone mineralization, leading to conditions such as rickets and decreased bone density[25]. Children with vitamin D deficiency were found to have a higher incidence of respiratory infections and weakened immune responses. Additionally, insufficient vitamin D levels were correlated with delayed physical growth, reduced muscle strength, and increased risk of skeletal deformities. Epidemiological data indicated that vitamin D deficiency is more common in regions with limited sunlight exposure, darker skin pigmentation, and inadequate dietary intake[27]. Seasonal variations also significantly influenced vitamin D levels, with lower concentrations observed during winter months.

Discussion. The results of this study confirm that vitamin D plays a critical role in both skeletal and extra-skeletal functions in children. Its involvement in calcium and phosphorus metabolism is essential for proper bone formation and growth. However, the presence of vitamin D receptors in various tissues suggests a broader biological role, particularly in immune regulation[28]. Vitamin D deficiency contributes to increased susceptibility to infections due to its role in modulating both innate and adaptive immune responses. It enhances the production of antimicrobial peptides and regulates inflammatory processes, thereby supporting immune defense mechanism[29]s. Furthermore, the high global prevalence of vitamin D deficiency highlights the need for effective preventive strategies[. Public health interventions, including food fortification, supplementation programs, and awareness of adequate sunlight exposure, are essential to address this issue. Despite the growing body of evidence, further large-scale clinical studies are required to fully understand the long-term effects of vitamin D on pediatric health and its role in preventing chronic diseases.

Conclusion. Vitamin D is an essential biological regulator involved in multiple physiological systems, including skeletal health, muscle function, and immune response. Its deficiency is associated with weakened bone structure, increased risk of falls and fractures, and the development of various chronic diseases. Therefore, maintaining adequate vitamin D levels through proper sunlight exposure, balanced nutrition, and supplementation when necessary is crucial for overall health and disease prevention.

References

1. Holick, M. F. (2006). High prevalence of vitamin D inadequacy and implications for health. *Mayo Clinic Proceedings*, 81(3), 353–373.
2. Wagner, C. L., & Greer, F. R. (2008). Prevention of rickets and vitamin D deficiency in infants and children. *Pediatrics*, 122(5), 1142–1152.
3. Misra, M., Pacaud, D., Petryk, A., Collett-Solberg, P. F., & Kappy, M. (2008). Vitamin D deficiency in children and its management. *Pediatrics*, 122(2), 398–417.
4. Rosen, C. J. (2011). Clinical practice. Vitamin D insufficiency. *New England Journal of Medicine*, 364(3), 248–254.
5. DeLuca, H. F. (2004). Overview of general physiologic features and functions of vitamin D. *American Journal of Clinical Nutrition*, 80(6), 1689S–1696S.
6. Holick, M. F., & Chen, T. C. (2008). Vitamin D deficiency: A worldwide problem. *American Journal of Clinical Nutrition*, 87(4), 1080S–1086S.
7. Norman, A. W. (2008). From vitamin D to hormone D: Fundamentals of the vitamin D endocrine system. *American Journal of Clinical Nutrition*, 88(2), 491S–499S.
8. Christakos, S., Ajibade, D. V., Dhawan, P., Fechner, A. J., & Mady, L. J. (2010). Vitamin D: Metabolism and mechanism of action. *Endocrinology and Metabolism Clinics*, 39(2), 243–253.
9. Jones, G. (2008). Pharmacokinetics of vitamin D toxicity. *American Journal of Clinical Nutrition*, 88(2), 582S–586S.
10. Lips, P. (2006). Vitamin D physiology. *Progress in Biophysics and Molecular Biology*, 92(1), 4–8.
11. Heaney, R. P. (2004). Functional indices of vitamin D status and ramifications of vitamin D deficiency. *American Journal of Clinical Nutrition*, 80(6), 1706S–1709S.
12. Garland, C. F., Garland, F. C., Gorham, E. D., et al. (2006). The role of vitamin D in cancer prevention. *American Journal of Public Health*, 96(2), 252–261.
13. Giovannucci, E. (2005). The epidemiology of vitamin D and cancer incidence and mortality. *Cancer Causes & Control*, 16(2), 83–95.