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VITAMIN D DEFICIENCY IN CHILDREN – EPIDEMIOLOGY, HEALTH CONSEQUENCES AND PREVENTION. A REVIEW OF THE LITERATURE

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ABSTRACT

For many years, a succession of scientific studies and publications have confirmed the significant impact vitamin D has on the functioning of the human body. It is responsible for a range of biological processes, acting as a multifunctional hormone or prohormone; it regulates the immune system, influences hormonal balance, calcium and phosphate metabolism, and even mental functioning.

In the case of the paediatric population, on which we focus our review, proper physical development and growth are of paramount importance. It is widely recognised that the aforementioned processes are essential for this.

Our review is based on numerous publications addressing the issue of vitamin D deficiency and its impact on child development. We describe the general problem, current statistics, the mechanism of synthesis and absorption of the active form of vitamin D, and the impact of deficiency on specific aspects of bodily function. The paper also includes current guidelines on vitamin D supplementation for the Polish population.

KEYWORDS

Vitamin D, Children, Deficiency, Supplementation, Paediatrics, 25(OH)D

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Introduction

1. The importance of vitamin D in a child's body

Vitamin D is a prohormone that plays a key role in regulating calcium and phosphorus metabolism and is a major factor in determining bone health during childhood and adolescence [1]. Vitamin D supplementation during infancy prevents rickets and osteomalacia, thereby increasing, amongst other things, bone mineral density [2].

2. Definition of deficiency

In recent decades, our understanding of the link between vitamin D levels and children's health has expanded significantly. Deficiency is generally defined as a condition in which the serum 25(OH)D concentration is below 50 nmol/l or 20 ng/ml [3].

3. The scale of the global deficiency problem

It is well known that vitamin D deficiency causes rickets in children and osteomalacia in adults. Following the discovery of vitamin D and the introduction of fortification in food, nutritional rickets virtually disappeared from developed countries. However, over the last two decades, a number of factors have led to the re-emergence of this condition, and the incidence is rising, particularly among non-Caucasian children [1, 4].

Studies have confirmed that a significant number of healthy children in Europe may be deficient in vitamin D, particularly those in high-risk groups (those with darker skin, those living in areas with limited sun exposure, and those with other medical conditions) [2].

Vitamin D levels vary across European countries, and these differences can sometimes be unexpected depending on the population studied. Depending on the specific study, 25(OH)D concentrations below 10 ng/ml are found in 2–30% of the European population, with this proportion reaching as high as 75% among elderly people in care homes and postmenopausal women with osteoporosis [5].

4. Aim of the study

This article discusses the key aspects of vitamin D deficiency in children in the light of the latest research and highlights the most significant health consequences of this condition.

Materials and Methods

This article is a review. The data used in the article was obtained from available peer-reviewed scientific articles and reports published in reputable journals and databases. We used online databases and journals such as PubMed, MDPI, International Journal of Pediatrics, Medycyna ogólna i Nauki o Zdrowiu, Journal of Pediatric Endocrinology and Metabolism, La Presse Medicale, The International Journal of Artificial Organs, Annals of Medicine, Cambridge University Press. The review selected clinical trials, review articles, guidelines and reports on Vitamin D deficiency in Children published between 2010 and 2025 in English and Polish.

Results (Review of the Literature)

1. Metabolism and physiological role of vitamin D

- **cutaneous synthesis**

There are two forms of vitamin D: cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2). Cholecalciferol comes from dietary sources (oily fish such as salmon and mackerel, animal liver, fish liver oils, eggs), but is also produced in the skin from 7-dehydrocholesterol (7-DHC or provitamin D3). When exposed to light, 7-DHC absorbs solar radiation (ultraviolet B or UVB rays, with a wavelength of 290–315 nm), which causes it to be converted into provitamin D3 [6].

The basal layer and the spinous layer of the epidermis have the greatest capacity for the synthesis of previtamin D3. Once formed, previtamin D3 undergoes isomerisation to form vitamin D3, which is then transported into the extracellular space and the capillaries of the dermis[1].

Children, and infants in particular, need less sunlight to produce sufficient amounts of vitamin D, due both to their larger body surface area relative to their height and their greater ability to produce vitamin D compared to older people. [1]

- metabolism in the liver and kidneys

Both cholecalciferol and ergocalciferol are biologically inactive, and their activation requires further hydroxylation reactions taking place in the liver and kidneys. In the liver, both forms of vitamin D are converted into the 25-hydroxylated compound (25(OH)D), which is then stored in the liver and adipose tissue. When required, it is then converted in the kidneys by the enzyme 1-alpha-hydroxylase into the biologically active form of vitamin D (1,25(OH)2D), under the control of the parathyroid glands. This form of vitamin D is also produced in other tissues, such as intestinal cells, smooth muscle cells of blood vessels, B lymphocytes, monocytes and dendritic cells [1,7].

- regulation of calcium metabolism

Calcitriol, referred to by many sources as a hormone, is primarily known for its key role in increasing the amount of calcium absorbed in the gastrointestinal tract and in promoting the mineralisation of osteoid tissue. It also stimulates calcium reabsorption in the renal tubules, and in the skeletal system, calcitriol interacts with VDR receptors in osteoblasts and works in conjunction with PTH to regulate bone turnover. Calcitriol not only increases the absorption of calcium and phosphorus in the intestines, but also inhibits PTH activity, thereby suppressing the proliferation of parathyroid cells and the secretion of their hormones [8].

2. Epidemiology of vitamin D deficiency in children

- Prevalence in different regions of the world / European and global data

Studies show that vitamin D deficiency is diagnosed in 56.8% of children in the Polish population. It is estimated that vitamin D deficiency ranges from 6.9% to 81.8% in European countries and from 2.0% to 87.5% in Asian countries [9,10].

- High-risk groups

Latitude, time of day and season significantly influence the production of cholecalciferol in the skin. Furthermore, melanin competes with 7-DHC for UVB photons, which reduces cholecalciferol production. In fact, people with darker skin tones require longer exposure to the sun to produce cholecalciferol. Older people also require longer exposure to the sun to produce the same amount of cholecalciferol, as the 7-DHC content in their skin is reduced by up to 70% compared to young adults [6, 11].

Other key factors contributing to vitamin D deficiency include spending time indoors and leading a sedentary lifestyle, which are linked to insufficient exposure to sunlight, air pollution, obesity and the use of sunscreen [9, 12].

Interestingly, taking anti-epileptic drugs (carbamazepine) and anticonvulsants (levetiracetam) leads to a reduction in vitamin D levels in the body[13].

Furthermore, the atrophy of the intestinal villi associated with coeliac disease leads to malabsorption of nutrients, reducing the absorption of vitamin D, calcium and magnesium [14].

3. Risk factors

- **limited exposure to sunlight**

The main cause of vitamin D deficiency in the general population (including children) is insufficient exposure to sunlight. According to various sources, endogenous production accounts for as much as 70–80% of the total amount of this vitamin circulating in the human body [15].

7-Dehydrocholesterol (an intermediate compound in the synthesis of cholesterol, which accumulates in the skin) undergoes non-enzymatic conversion to previtamin D under the influence of ultraviolet light. Within an hour, it undergoes a further reaction, resulting in the formation of cholecalciferol, which is absorbed into the bloodstream. In people living in temperate climates, the highest plasma concentrations of vitamin D are found at the end of summer, and the lowest at the end of winter. At latitudes north or south of the 40th parallel, the availability of UV light of sufficient intensity is very low during the winter months [16].

This applies primarily to people with darker skin, who, due to the higher melanin content in their skin, require longer exposure to sunlight to produce the same amount of cholecalciferol as people with fair skin [17].

A similar effect can be observed with the excessive use of sunscreen, which inhibits the skin's production of vitamin D by preventing sunlight from reaching the deeper layers of the skin [18].

It is only when the skin does not receive sufficient exposure to sunlight that it becomes necessary to obtain this vitamin from food.

- **a diet low in vitamin D**

Although vitamin D is found in many animal-based products, such as dairy products and oily fish, our body's requirement for this vitamin is mainly met through its own production in the skin when exposed to UV-B radiation [19].

- **obesity**

An extremely important yet often overlooked group of patients at risk of vitamin D deficiency are obese individuals, due to its sequestration in subcutaneous tissue. This is because it is stored in fat cells, which inhibits the transport of de novo synthesised vitamin D from the skin into the bloodstream.

The distribution of 25(OH)D to serum, muscle, fat and liver—compartments in which concentrations are elevated in obesity—is currently one of the most likely mechanisms underlying low vitamin D levels [20].

Some studies have shown a smaller increase in serum 25(OH)D levels in obese individuals than in those of normal weight in response to vitamin D supplementation. In some studies, this difference was as high as 30% [21,22].

Some researchers therefore suggest that the recommended dose of vitamin D should be based on body weight in order to account for differences in serum levels between obese and lean individuals [23].

It is worth noting that there has recently been a growing body of research and data supporting the hypothesis that vitamin D may play a role in the pathogenesis of obesity, rather than merely being a consequence of it. Some studies suggest that elevated levels of parathyroid hormone, caused by vitamin D deficiency, promote lipogenesis through an increased influx of calcium into adipocytes [24,25].

4. Clinical consequences

- **Skeletal effects**

The effect of vitamins, and particularly vitamin D, on vitality and normal development has been the subject of research for over a century. One of the earliest experiments was conducted in 1905 by Cornelius Adrianus Pekelharing, in which he discovered that animals fed purified proteins/carbohydrates, fats and water would only develop properly if small amounts of milk were introduced into their diet. This led to the hypothesis that milk contains some unknown substance which, in very small quantities, is essential for proper growth and development. Researchers, of course, could not yet have known that one of the essential substances at that time was vitamin D [26].

It was not until 1919 that Sir Edward Mellanby discovered vitamin D and its role in the pathogenesis of rickets. The researcher correctly observed a link between the lack of exposure to sunlight in 20th-century England and the preventive effects of cod liver oil [27,28].

- **rickets**

The regulation of the calcium-phosphate pathway by vitamin D is a complex process involving many organs, signalling molecules and receptors in the human body. It is worth noting that this substance exists in two forms: Vitamin D2 (ergocalciferol), produced in plants, and Vitamin D3 (cholecalciferol), which is initially synthesised by the skin. The synthesis pathway begins in the dermis, where, under the influence of UV radiation, 7-dehydrocholesterol is converted into cholecalciferol. Subsequently, 25(OH)D3 is formed in

the liver, followed by 1,25(OH)2 via the renal pathway. The active form of vitamin D influences the release of osteoclasts and the absorption of calcium and phosphate in the intestine [29].

Rickets has been defined as a failure of mineralisation in newly forming bone tissue, which is associated with growth disturbances due to widening of the growth plate and concavity of the metaphysis, leading to curvature of the long bones and characteristic widening of the rib ends [30].

The most common form is, of course, so-called nutritional rickets. In addition to this, there is also vitamin D-dependent rickets, which is associated with hereditary gene mutations that affect the conversion of vitamin D into its active form in the kidneys [31].

Bone formation is a vital process in which vitamin D plays a key role, but we must not forget that bone healing is also linked to it. Research into the impact of vitamin D deficiency on the risk of fractures has been ongoing for some time. In one of the recommendations, a group of experts stated that children with radiologically confirmed rickets have an increased risk of fractures, whereas children with simple deficiency do not [32].

Similar findings are reported in a 2014 study which assessed the relationship between vitamin D levels in 100 children who had suffered a fracture and those who were healthy. That study found no link between vitamin D deficiency and the risk of fractures [33].

On the other hand, a 2017 study found that children with fractures had significantly lower levels of 25(OH)D compared with healthy children (14.5 ng/ml vs 21.3 ng/ml) [34].

A similar study from 2016 found a higher prevalence of vitamin D deficiency in children with forearm fractures [35].

Given the conflicting results of many studies, it is essential to carry out further, more detailed analyses.

- **Non-skeletal effects**
- **childhood autism**

Autism spectrum disorders (ASD) are neurodevelopmental disorders characterised by impaired social interaction with peers, repetitive behaviours and specific interests. It is, of course, a multifactorial condition that is the subject of a vast amount of scientific research. Naturally, some of these studies seek to establish a link between low vitamin D levels in children and the development of childhood autism. One study found that 223 genes involved in the pathogenesis of ASD are vitamin D-sensitive genes. Another study found lower levels of vitamin D in children with ASD [36].

Another study aimed at determining the link between autism spectrum disorder (ASD) and vitamin D levels in children was published in 2020 by Esma Şengenç. The researchers measured serum 25-hydroxyvitamin D levels in 1,529 patients with ASD aged between 3 and 18 years. It was noted that the study accounted for the absence of other chronic conditions, the season, gender and age of the participants. Vitamin D deficiency was found in approximately 95% of all patients, with 25-OHD concentrations in patients aged 11–18 being significantly lower than in those under 11 years of age. This study suggests a link between vitamin D and ASD in children [37].

Attempts have also been made to treat ASD through vitamin D supplementation. Some studies have shown a significant reduction in symptom severity, such as the one conducted on a small group of 43 children (7 girls and 36 boys) [38].

On the other hand, there are many studies that have found no significant difference between vitamin D therapy and a placebo. The researchers note that supplementation was beneficial only in cases of hyperactivity [39].

The discrepancies in the research only reinforce our belief that this topic requires further study

- **effect on the immune system**

Another widely discussed function of vitamin D in the human body is, of course, its effect on the immune system. Calcitriol, the active form of vitamin D, reduces oxidative stress mainly by suppressing inflammatory cytokines. Its immunomodulatory action involves the activation of immune system cells, such as T and B lymphocytes, as well as macrophages and dendritic cells [40].

Chronic hypovitaminosis appears to increase susceptibility to infection, particularly to intracellular bacteria such as tuberculosis and respiratory viruses [41].

One of the most recent studies on this topic was a retrospective analysis conducted and published in 2020 by Antonio D'Avolio's team. In this cohort, significantly lower levels of 25(OH)D (p=0.004) were found in patients with a positive SARS-CoV-2 test result (median 11.1 ng/ml) compared with those with a negative result (24.6 ng/ml). However, the researchers point to the need for further studies involving a larger number of participants [42].

Another RCT was conducted by Camargo and involved administering 300 IU of vitamin D daily for three months to 247 Mongolian children. The incidence of respiratory infections was then assessed in the treatment and control groups. Interestingly, supplementation resulted in a reduction in the incidence of upper respiratory tract infections.

Results: At baseline, the median serum 25(OH)D level was 7 ng/mL (interquartile range: 5-10 ng/mL). At the end of the trial, follow-up was 99% (n = 244), and the median 25(OH)D levels of children in the control versus vitamin D groups was significantly different (7 vs 19 ng/mL; P <.001). Compared with controls, children receiving vitamin D reported significantly fewer ARIs during the study period (mean: 0.80 vs 0.45; P =.047), with a rate ratio of 0.52 (95% confidence interval: 0.31-0.89). Adjusting for age, gender, and history of wheezing, vitamin D continued to halve the risk of ARI (rate ratio: 0.50 [95% confidence interval: 0.28-0.88]). Similar results were found among children either below or above the median 25(OH)D level at baseline (rate ratio: 0.41 vs 0.57; P(interaction) =.27) [43].

Similar results were obtained when 4000 IU of vitamin D was administered to immunocompromised patients in Sweden. The study involved 140 patients, with a control group also included, and vitamin D was administered over a period of one year. The overall infectious score was significantly reduced for patients allocated to the vitamin D group (202 points) compared with the placebo group (249 points; adjusted relative score 0.771, 95% CI 0.604 to 0.985, p=0.04) [44].

A double-blind trial conducted by Urashima showed that children supplemented with 1200 IU of vitamin D per day had a significantly lower incidence of influenza A (18.6%) compared with the placebo group (10.8%). In this study, vitamin D supplementation was significantly effective in children with asthma [45].

- **chronic conditions**

Vitamin D deficiency may also be associated with many autoimmune diseases, including type 1 diabetes. Studies show that in children with type 1 diabetes, generalised 25OHD deficiency affects metabolic status and glycaemic homeostasis, whilst vitamin D supplementation improves glycaemic control. Observational studies have shown that improving vitamin D status and supplementing with vitamin D from the first days of a child's life reduces the risk of developing this subtype of diabetes. [1]

Recent studies show that serum vitamin D levels are lower in patients with atopic dermatitis. Atopic dermatitis is a chronic, inflammatory skin condition that affects many children worldwide. It is usually caused by a compromised skin barrier and an abnormal immune response. Vitamin D supplementation may offer therapeutic benefits for children with AD [46].

5. Diagnosis of vitamin D deficiency

- **Blood vitamin D test / reference values**

Vitamin D levels are measured based on the concentration of 25(OH)D in the blood serum. If the 25(OH)D concentration is below 75 nmol/l (or 30 ng/ml), this is considered a vitamin D deficiency. When the concentration is below 25 or 30 nmol/l (or 10/12 ng/ml), this is considered a severe vitamin D deficiency [9]. The critical threshold mentioned in the previous paragraph is 50 nmol/l, as proposed by the Institute of Medicine (United States) and the Endocrine Society's Vitamin D Working Group [9].

6. Prevention and supplementation

Epidemiological studies agree that vitamin D deficiency is widespread among children worldwide. In light of numerous studies, including those cited above, which demonstrate the adverse effects of vitamin D deficiency on the risk of various diseases, many scientific societies recommend daily supplementation at a dose appropriate to the child's age [47,48].

- **Recommendations for supplementation in children**

Current recommendations for vitamin D supplementation in Poland are based on the use of cholecalciferol or, in specific medical cases, calcifediol. General guidelines advise that the dose of cholecalciferol should be tailored to the individual, taking into account the child's age, body weight, exposure to sunlight, dietary habits and lifestyle.

- **Vitamin D intake in different age groups**

The latest guidelines on vitamin D supplementation doses are as follows:

a) Full-term newborns and infants: Ages 0–6 months: 400 IU/day (10 µg/day) of cholecalciferol from the first days of life, regardless of feeding method. Age 6–12 months: 400–600 IU/day (10–15 µg/day) of cholecalciferol, depending on the daily amount of vitamin D consumed with meals. The use of calcifediol is not recommended in full-term newborns and healthy infants.

b) Children (1–10 years): In healthy children aged 1–3 years, supplementation should be based on the administration of cholecalciferol at a daily dose of 600 IU (15 µg/day). Due to age-related restrictions on sun exposure, supplementation is recommended throughout the year. In healthy children aged 4–10 years who sunbathe with their forearms and legs exposed for 15–30 minutes between 10:00 and 15:00 without sunscreen, from May to the end of September, cholecalciferol supplementation is not necessary, although it is still recommended and safe. If these guidelines are not met in healthy children aged 4–10 years, supplementation with cholecalciferol at a dose of 600–1000 IU/day (15–25 µg/day) is recommended throughout the year, depending on body weight and dietary vitamin D intake. Calcifediol is not recommended for healthy children aged 1–10 years.

c) Adolescents (11–18 years): In healthy adolescents, cholecalciferol should be the first-choice supplement for the prevention of vitamin D deficiency, followed by calcifediol. In healthy adolescents who sunbathe with their forearms and legs exposed for 30–45 minutes between 10:00 and 15:00 without sunscreen, from May to the end of September, cholecalciferol supplementation is not necessary, although it is still recommended and safe. If the above guidelines are not met, supplementation with cholecalciferol at a dose of 1000–2000 IU/day (25–50 µg/day) is recommended throughout the year, depending on body weight and dietary vitamin D intake. If the above guidelines are not met, alternative prophylaxis based on the intake of calcifediol at a daily dose of 10 µg (oral solution) throughout the year is recommended, and a follow-up test of serum 25(OH)D concentration should be performed 6–8 days after the start of supplementation.

d) Supplementation in groups at risk of vitamin D deficiency: In patients at risk of vitamin D deficiency, supplementation with cholecalciferol or calcifediol should be initiated and continued under the supervision of serum 25(OH)D levels to achieve and maintain optimal levels of >30–50 ng/ml. If assessment of serum 25(OH)D levels is not possible in at-risk groups, cholecalciferol dosing should be guided by the guidelines for the general population, using the maximum doses for the relevant age group. Alternatively, for prophylaxis, calcifediol at a daily dose of 10 µg (oral solution). Overweight and obesity require particular attention, as this condition usually requires a double dose of cholecalciferol compared to the doses recommended for peers of normal weight. In obese individuals, calcifediol at a daily dose of 10 µg (oral solution) may be considered as an alternative, second-line option for prevention. Obesity in children and adolescents is defined as a BMI >90th percentile for age and reference sex; in adults and the elderly, obesity is defined as a BMI >30 kg/m² [49,50].

7. Conclusions

Vitamin D deficiency is a common paediatric problem

Vitamin D deficiency affects a significant number of children and may be associated with serious health problems in childhood, and vitamin D may play a potential role in their aetiopathogenesis. Its levels are crucial for bone health in infants, children and adolescents, including bone mineralisation disorders, rickets (bone deformities, growth disorders), and a possible link to an increased risk of fractures (research findings are inconclusive). Studies also show that low vitamin D levels may be associated with autism spectrum disorders (ASD), increased susceptibility to infections (e.g. respiratory viruses, tuberculosis), a weakened immune response, or a higher risk of infection in cases of chronic deficiency.

Early diagnosis and supplementation are crucial

Diagnosing vitamin D deficiency is often difficult based on clinical symptoms, which is why supplementation is recommended from the very first days of life as a preventive measure. One of the current challenges is achieving and maintaining optimal serum 25(OH)D levels, which benefit all tissues in the body. Cholecalciferol (vitamin D3) is the primary form of supplementation in all age groups. Calcifediol is used less frequently — mainly in adolescents as an alternative.

Vitamin D doses increase with age and depend on diet and sun exposure, which can partially replace supplementation, but a lack of it means that vitamin D must be taken all year round. Newborns and infants from 0 to 6 months: 400 IU (10 µg) daily from the first days of life — regardless of feeding method; from 6 to 12 months: 400–600 IU (10–15 µg) daily — depending on the amount of vitamin D in the diet. Children aged 1 to 3 years: 600 IU (15 µg) daily throughout the year (in the case of limited sun exposure). Children aged 4 to 10: if the child has regular exposure to sunlight (15–30 minutes a day from May to September): supplementation is not necessary, but is still safe and recommended; if there is no such exposure: 600–1000 IU (15–25 µg) a day throughout the year. For adolescents with adequate sun exposure (30–45 minutes a day from May to September): supplementation is not necessary, but is still recommended and safe; if there is no exposure: 1000–2000 IU (25–50 µg) a day throughout the year.

Further research is needed

A key priority is to raise public awareness of the role of vitamin D and the consequences of its deficiency, as well as to educate parents (particularly regarding supplementation for children from the very first days of life). One of the most important tasks is regular supplementation (especially during the autumn–winter period or when there is little exposure to sunlight), including the individual adjustment of doses (age, body weight, diet) and paying greater attention to groups at risk of deficiency (infants, children, adolescents, people with little exposure to sunlight). Increasing the intake of foods rich in vitamin D (e.g. oily fish, eggs) or fortifying foods (e.g. milk, margarine) with vitamin D, together with supplementation, can ensure adequate vitamin D levels in the blood.

Furthermore, more frequent testing of 25(OH)D levels in at-risk groups, conducting further research and ensuring compliance with medical advice, implementing public health programmes (e.g. free supplementation for children), and supporting doctors in preventive measures will have the greatest impact on the nature of the problem.

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