


# Vitamin D supplementation in children with obesity: implications for clinical practice and current evidence

## Suplementacja witaminy D u dzieci z otyłością: implikacje kliniczne i aktualny stan wiedzy

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### Abstract

**Introduction:** Childhood obesity is associated with an increased risk of vitamin D deficiency due to volumetric dilution, adipose sequestration, and altered metabolism. Vitamin D plays a crucial role in bone health, immune function, and metabolic regulation; however, optimal supplementation strategies for children with obesity remain unclear. Current paediatric guidelines do not provide specific recommendations for this population. This review evaluates the prevalence of vitamin D deficiency in children with obesity and summarises evidence on effective supplementation regimens. **Methods:** A structured narrative review of literature up to February 2026 was conducted using PubMed and Google Scholar. Clinical trials involving children with obesity aged 5–20 years, with clearly defined methodology and reported 25(OH)D concentrations, were included. **Results:** Children with obesity demonstrate a blunted response to vitamin D supplementation compared with normal-weight peers. Standard doses (400–2,000 IU/day) frequently fail to achieve serum 25(OH)D  $\geq 30$  ng/mL. Higher doses ( $\geq 4,000$  IU/day or equivalent weekly/monthly regimens) are more effective in normalising vitamin D status. No significant adverse effects were reported across included studies. **Conclusions:** Children with obesity may require individualised, higher-dose vitamin D supplementation to achieve adequate serum 25(OH)D levels. Further research is needed to establish evidence-based dosing guidelines and to evaluate long-term safety in this population.

**Keywords:** vitamin D deficiency, supplementation, 25-hydroxyvitamin D, paediatric obesity

### Streszczenie

**Wprowadzenie:** Otyłość dziecięca wiąże się ze zwiększonym ryzykiem niedoboru witaminy D z powodu rozcieńczenia objętościowego, sekwestracji w tkance tłuszczowej oraz zaburzeń metabolicznych. Witamina D odgrywa kluczową rolę w zdrowiu kości, funkcjonowaniu układu odpornościowego i regulacji metabolicznej, jednak optymalne strategie suplementacji u dzieci z otyłością pozostają niejasne. Aktualne wytyczne pediatryczne nie zawierają szczegółowych zaleceń dla tej populacji. Celem niniejszego przeglądu jest ocena częstości występowania niedoboru witaminy D u dzieci z otyłością oraz podsumowanie dowodów dotyczących schematów suplementacji. **Metody:** Przeprowadzono strukturyzowany przegląd narracyjny literatury w bazach PubMed i Google Scholar do lutego 2026 roku. Włączono badania kliniczne z udziałem dzieci z otyłością w wieku 5–20 lat, o jasno zdefiniowanej metodologii i raportowanych stężeniach 25(OH)D. **Wyniki:** Dzieci z otyłością wykazują osłabioną odpowiedź na suplementację witaminy D w porównaniu z rówieśnikami z prawidłową masą ciała. Standardowe dawki (400–2000 j.m./dobę) często nie pozwalają uzyskać stężenia 25(OH)D  $\geq 30$  ng/ml. Wyższe dawki ( $\geq 4000$  j.m./dobę lub równoważne dawki tygodniowe/miesięczne) są skuteczniejsze w normalizacji poziomu witaminy D. W żadnym z włączonych badań nie odnotowano istotnych działań niepożądanych. **Wnioski:** Dzieci z otyłością mogą wymagać zindywidualizowanej suplementacji witaminy D w wyższych dawkach w celu osiągnięcia odpowiednich stężeń 25(OH)D w surowicy. Konieczne są dalsze badania w celu opracowania wytycznych opartych na dowodach naukowych, uwzględniających optymalne dawkowanie i długoterminowe bezpieczeństwo w tej populacji.

**Słowa kluczowe:** niedobór witaminy D, suplementacja, 25-hydroksywitamina D, otyłość dziecięca

## BACKGROUND AND INTRODUCTION

Childhood obesity has emerged as a major global public health challenge. The World Health Organization reports a dramatic increase in overweight and obesity among children and adolescents over recent decades, affecting millions worldwide across both developed and developing countries. As of 2022, approximately 20% of individuals aged 5–19 are classified as overweight, including obesity<sup>(1)</sup>. Paediatric obesity is associated with numerous short- and long-term health consequences, including metabolic, cardiovascular, and endocrine disturbances<sup>(2,3)</sup>.

Among these, vitamin D deficiency appears to be particularly prevalent. Depending on the population studied and the definition of deficiency applied, prevalence estimates range from 40% to over 60% in most cohorts, with the highest rates observed in urban areas and during seasons with limited sunlight exposure<sup>(4–6)</sup>. In certain populations, particularly in regions with high skin pigmentation or limited sun exposure, the prevalence may exceed 90%<sup>(7)</sup>. Skin pigmentation itself is a key determinant of vitamin D status. Studies have shown that among children with obesity, deficiency affects up to 71–87% of African American youth and 44–52% of Latino youth, with prevalence rates significantly higher in autumn and winter compared with spring and summer<sup>(8)</sup>. Even among Polish children, a higher melanin index is associated with lower 25(OH)D concentrations, particularly during autumn months<sup>(9)</sup>.

The clinical consequences of vitamin D deficiency in children with obesity can be substantial, yet often remain underrecognised. A complex adaptive response may modify the clinical presentation, making symptoms subtle and frequently overlooked, which complicates accurate prevalence assessment. Adolescents with severe deficiency may present with vague, nonspecific manifestations such as pain in weight-bearing joints, back, thighs, or calves, difficulty walking or climbing stairs, and muscle cramps. This adaptive process involves hormonal shifts which, over time, may adversely affect bone mineralisation, potentially leading to osteopenia and osteoporosis<sup>(10)</sup>. Beyond these skeletal effects, vitamin D plays a crucial role in immune regulation, glucose metabolism, and inflammatory pathways<sup>(3,11,12)</sup>. Emerging evidence suggests that suboptimal vitamin D status in children with obesity may contribute to insulin resistance, dyslipidaemia, and low-grade chronic inflammation, potentially amplifying cardiometabolic risk<sup>(2,13)</sup>.

The pathophysiology underlying vitamin D deficiency in children with obesity is multifactorial. A well-documented inverse relationship exists between body mass index (BMI) and serum 25-hydroxyvitamin D [25(OH)D] concentrations in children, establishing obesity as a major risk factor for vitamin D deficiency and lower circulating levels<sup>(7,14,15)</sup>.

Several mechanisms have been proposed to explain lower 25(OH)D levels in children with obesity, including volumetric dilution, sequestration in adipose tissue, altered

hepatic 25-hydroxylation, and potential differences in vitamin D receptor expression<sup>(2,4,15,16)</sup>. Importantly, vitamin D status in children with obesity is shaped not only by altered distribution and metabolism but also by environmental and lifestyle determinants. Limited outdoor activity, increased screen time, and reduced sun exposure – frequently observed in this population – may significantly decrease endogenous vitamin D synthesis. Dietary patterns characterised by low intake of vitamin D-rich foods, combined with socioeconomic constraints affecting food quality, fortification exposure, and access to supplementation, may further contribute to hypovitaminosis D. Additionally, children with darker skin pigmentation are at increased risk due to reduced cutaneous synthesis, and this risk may be further amplified in populations facing socioeconomic disadvantage, where limited access to safe outdoor spaces, nutrient-dense foods, and healthcare services may compound existing vulnerabilities<sup>(10,17)</sup>. These interacting biological and social determinants underscore the multifactorial nature of vitamin D deficiency in this population and highlight the need for individualised strategies when addressing vitamin D deficiency in children with obesity.

Although standard vitamin D supplementation guidelines exist for the general paediatric population (e.g. American Academy of Pediatrics, European Society for Paediatric Gastroenterology Hepatology and Nutrition, Institute of Medicine), uncertainty remains regarding optimal dosing strategies in children with obesity. Due to the lipophilic nature of vitamin D, its sequestration in adipose tissue, and an altered volume of distribution, standard supplementation doses may be insufficient<sup>(2,4,7)</sup>.

Therefore, the aim of this review is to provide a comprehensive overview of the prevalence of vitamin D deficiency in children with obesity and to critically analyse current evidence regarding supplementation strategies, dosing considerations, and existing clinical recommendations.

## RESEARCH MATERIALS AND METHODS

This study was conducted as a structured narrative review of current literature on vitamin D deficiency and supplementation in obese paediatric populations. A literature search was performed using PubMed and Google Scholar databases up to March 2026. The following keywords were applied: “vitamin D supplementation”, “vitamin D deficiency”, “paediatric obesity”, “obese children”, and “25(OH)D”. Boolean operators were used to refine the search strategy. Studies were included if they involved children or adolescents aged 5–20 years, addressed obesity defined by BMI criteria, and evaluated vitamin D status and supplementation strategies. In the included studies, obesity was defined using various anthropometric criteria, predominantly body mass index (BMI) percentiles or Z-scores according to age- and sex-specific reference charts (including World Health Organization growth standards, national reference centile charts, and other established paediatric criteria), with

cut-offs ranging from  $\geq 85^{\text{th}}$  percentile for overweight to  $\geq 95^{\text{th}}$  percentile or  $\geq 2$  standard deviations above the mean for obesity. Only studies with clearly defined methodology and reported baseline and post-treatment serum 25(OH)D concentrations were included in the primary analysis.

Studies with unclear methodology, inconsistent reporting, or missing outcome data were excluded, as were studies conducted exclusively in adults, animal studies, case reports, editorials, and non-English publications. The analysis focused primarily on clinical trials, including both randomised and non-randomised interventions. Other types of publications, such as narrative reviews or guidelines, were used in the introduction or discussion to provide background and context but were not included in the quantitative synthesis.

## LITERATURE REVIEW

The following section presents a synthesis of clinical trials assessing vitamin D supplementation in paediatric populations with obesity. Studies included in this review employed varying dosing regimens, durations, and schedules, allowing for a comparative evaluation of supplementation strategies and their effectiveness in achieving sufficient serum 25(OH)D concentrations of  $\geq 30$  ng/mL (Tab. 1).

The included intervention studies demonstrated substantial heterogeneity in dosing regimens, encompassing daily, weekly, and monthly supplementation strategies. Baseline 25(OH)D concentrations were generally within the deficient or insufficient range across cohorts. Treatment response varied considerably depending on dose and supplementation schedule.

Standard paediatric doses (400–800 IU/day), as well as moderate doses such as 1,000–2,000 IU/day, were frequently insufficient to achieve serum 25(OH)D concentrations  $\geq 30$  ng/mL in obese participants. In several trials, a substantial proportion of children remained below the sufficiency threshold despite supplementation. Even daily doses of 2,000 IU did not consistently normalise vitamin D status<sup>(2,5,18–20,23,28)</sup>.

Higher daily doses, particularly  $\geq 4,000$  IU/day,  $\geq 25,000$  IU/week, and  $\geq 100,000$  IU/month, were more likely to achieve serum concentrations above 30 ng/mL. Studies employing such dosing strategies reported higher normalisation rates compared with standard regimens<sup>(7,22–27,29)</sup>.

A dose–response relationship appears to emerge from the available data, with higher daily doses consistently associated with greater increases in serum 25(OH)D concentrations.

Notably, considerable heterogeneity exists across studies in dosing regimens, duration of supplementation, and target serum thresholds, complicating direct comparisons and limiting the development of definitive clinical recommendations.

The majority of studies were characterised by relatively short follow-up periods, restricting conclusions regarding long-term maintenance of vitamin D sufficiency.

## DISCUSSION

The present review demonstrates that children with obesity frequently exhibit a blunted response to vitamin D supplementation compared with their normal-weight peers<sup>(7,14,16,29)</sup>. Despite adherence to standard paediatric dosing regimens, serum 25(OH)D concentrations often remained below sufficiency thresholds, indicating that increased adiposity affects vitamin D bioavailability and storage. This pattern was observed consistently across multiple intervention studies employing daily, weekly, and monthly supplementation strategies<sup>(2,5,18–20,23–28)</sup>.

Existing clinical recommendations reflect a growing recognition that children with obesity require higher vitamin D doses than their normal-weight peers. Guidance across professional societies and expert consensus varies, encompassing both preventive and therapeutic approaches: some recommend 1,000–1,500 IU daily during periods of limited sun exposure, while the American Academy of Pediatrics advises 5,000 IU daily until stores are replenished in cases of documented deficiency, and the Endocrine Society suggests 2,000 IU daily or 50,000 IU weekly for at least six weeks. Interventional studies support these higher thresholds, demonstrating that doses of around 4,000 IU daily, or equivalent weekly or monthly regimens, effectively restore sufficiency in obese adolescents<sup>(15,30)</sup>. Collectively, these observations support the view that children with obesity typically require approximately double the vitamin D dose recommended for age-matched peers of normal weight.

Obesity alters vitamin D metabolism through increased sequestration of this lipophilic vitamin in adipose tissue, as well as through potential alterations in hepatic hydroxylation and other metabolic pathways, ultimately leading to lower circulating 25(OH)D concentrations<sup>(2,4,15,16)</sup>. Reduced vitamin D availability in children with obesity has been associated with disturbances in calcium and bone homeostasis, impaired immune function, and metabolic dysregulation<sup>(2,3,11–13)</sup>. These pathophysiological mechanisms further underscore the clinical importance of maintaining adequate vitamin D status in this at-risk population.

From a clinical perspective, paediatricians should recognise that children with obesity may require individualised vitamin D dosing strategies to achieve and maintain adequate serum 25(OH)D concentrations. Sustained daily supplementation appears to be as effective as weekly or monthly dosing regimens, although patient adherence and treatment compliance should be taken into consideration. Available evidence suggests that doses up to 4,000 IU/day are generally well tolerated; however, long-term safety data in obese paediatric populations remain limited. Regular monitoring of serum 25(OH)D concentrations, particularly in children receiving higher doses, may help guide individualised treatment and improve clinical outcomes.

From a public health perspective, optimising vitamin D screening and supplementation strategies among children with obesity may represent a cost-effective and scalable

Study	Intervention sample size (n)	Vitamin D dose	Duration	Baseline 25(OH)D (ng/mL)	Post-treatment 25(OH)D (ng/mL)
Asghari et al. (2021) <sup>(18)</sup>	378	A: 600 IU/d B: 1,000 IU/d C: 2,000 IU/d	12 months	11.5–12.2	A: 23.1 B: 25.6 C: 28.6
Brzeziński et al. (2020) <sup>(2)</sup>	85	1,200 IU/d	26 weeks	19.35	24.99
Javed et al. (2015) <sup>(19)</sup>	47	A: 400 IU/d B: 2,000 IU/d	12 weeks	24.0	A: 24.8 B: 27.1
Rajakumar et al. (2020) <sup>(20)</sup>	156	A: 600 IU/d B: 1,000 IU/d C: 2,000 IU/d	6 months	14.3	A: 23.8 B: 27.3 C: 30.1
Nader et al. (2014) <sup>(21)</sup>	44	2,000 IU/d	12 weeks	25.8	31.8
Setia et al. (2025) <sup>(22)</sup>	68	A: 4,000 IU/d B: 6,000 IU/d	12 weeks	9.4	A: 31.2 B: 40.2
Masoumi-Moghaddam et al. (2021) <sup>(7)</sup>	123	50,000 IU/week	12 weeks	20.15	49.8
Talib et al. (2016) <sup>(23)</sup>	183	A: 50,000 IU/week B: 5,000 IU/d C: 1,000 IU/d	8 weeks	13.7	A: 38.6 B: 34.7 C: 19.9
Kelishadi et al. (2014) <sup>(24)</sup>	21	25,000 IU/week	12 weeks	18.3	32.0
Samaranayake et al. (2020) <sup>(5)</sup>	52	A: 50,000 IU/week B: 2,500 IU/week	24 weeks	14.92	A: 18.34 B: 15.26
Sethuraman et al. (2018) <sup>(25)</sup>	29	50,000 IU/week	12 weeks	12.1	32.0
Rostampour et al. (2020) <sup>(26)</sup>	53	50,000 IU/week for 2 months, then 1,000 IU/d for 3 months	5 months	13.51	46.16
Javed et al. (2016) <sup>(27)</sup>	19	100,000 IU/month	3 months	22.36	34.76
Varshney et al. (2019) <sup>(28)</sup>	189	A: 120,000 IU/month B: 400 IU/d	12 months	8.36	A: 26.89 B: 13.14

Note: For consistency, all values originally reported in nmol/L have been converted to ng/mL using the conversion factor of 2.5 (nmol/L ÷ 2.5 = ng/mL).

Tab. 1. Characteristics of clinical trials assessing vitamin D supplementation in obese paediatric populations. The intervention sample size refers to participants receiving active vitamin D supplementation; control or placebo groups are not included. Daily, weekly and monthly regimens are presented, together with trial duration and baseline and post-treatment serum 25(OH)D concentrations

intervention to reduce long-term metabolic, skeletal, and immunological complications at the population level. This issue may be particularly relevant in Central and Eastern Europe, where pronounced seasonal variation in sunlight exposure and limited winter UVB availability may further increase the risk of vitamin D deficiency in children with obesity. Several limitations should be acknowledged when interpreting the findings of this review. Considerable heterogeneity exists across studies in terms of dosing regimens, supplementation duration, baseline vitamin D status, and target serum 25(OH)D thresholds, complicating direct comparisons between trials. In addition, many studies included relatively small sample sizes and short follow-up periods, limiting the ability to assess long-term maintenance of vitamin D sufficiency. These methodological differences also restrict conclusions regarding the long-term safety of higher supplementation doses in children with obesity. Although none of the reviewed studies reported clinically significant adverse effects, vitamin D toxicity remains a theoretical concern when high doses are used over prolonged periods. Toxicity is generally associated with excessive serum 25(OH)D concentrations, typically above 150 ng/mL, and is primarily mediated through hypercalcaemia. Potential clinical manifestations include nausea, vomiting, polyuria, dehydration, nephrocalcinosis, and renal

dysfunction. However, no such complications were observed in the intervention studies analysed in this review. Finally, relatively few trials have directly investigated optimal dosing strategies specifically tailored to obese paediatric populations, highlighting a critical gap in the literature and underscoring the need for larger, well-designed long-term studies.

## CONCLUSIONS

Vitamin D deficiency is highly prevalent in children with obesity and should be regarded as a routine clinical concern rather than an incidental finding. This population not only presents with lower baseline 25(OH)D concentrations but also exhibits a reduced response to standard supplementation regimens, likely due to altered vitamin D distribution and metabolism. A key clinical implication of the available evidence is that standard paediatric vitamin D doses are often inadequate in children with obesity. Multiple studies consistently indicate that higher doses may be required to achieve and maintain sufficient serum 25(OH)D levels. Therefore, a “one-size-fits-all” approach to supplementation should be avoided in this group. From a practical perspective, clinicians should adopt an individualised strategy that includes assessment of baseline vitamin D status, adjustment of dosing according to body

mass, and regular monitoring of serum 25(OH)D concentrations. Attention should also be given to modifiable factors such as sun exposure, diet, and physical activity, which may further influence vitamin D status.

Optimising vitamin D levels in children with obesity may have implications beyond bone health, potentially supporting metabolic and immune function. Given the growing prevalence of paediatric obesity, incorporating tailored vitamin D management into routine care may represent a simple yet impactful intervention.

Further high-quality, long-term studies are needed to define optimal dosing strategies and ensure safety. However, current evidence already supports a more proactive and individualised approach in clinical practice.

### Conflict of interest

*The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.*

### Author contribution

*Original concept of study; collection, recording and/or compilation of data; writing of manuscript: ZMJ; analysis and interpretation of data; critical review of manuscript; final approval of manuscript: ZMJ, LAP.*

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