

Review

The Role of Vitamin D in Pediatric Respiratory Diseases

Michele Miraglia del Giudice, Angela Klain, Giulio Dinardo, Salvatore Cascone, Carolina Grella, Nicola Montinaro, Cristiana Indolfi

Department of Woman, Child and General and Specialized Surgery, University of Campania 'Luigi Vanvitelli', Naples, Italy

ABSTRACT

Vitamin D is a pleiotropic secosteroid hormone involved in calcium-phosphate homeostasis, epithelial barrier integrity, antimicrobial defense, and adaptive immune regulation. Pediatric observational studies associate low serum 25-hydroxyvitamin D [25(OH)D] with respiratory infections, recurrent wheezing, asthma severity, impaired lung function, and reduced corticosteroid response, but the strength and consistency of these associations vary according to age, phenotype, baseline vitamin D status, comorbidities, and study design. Experimental evidence supports plausible effects on antimicrobial peptides, T-cell balance, airway smooth muscle, and glucocorticoid pathways. Interventional findings in selected trials in deficient or infection-prone children report fewer acute respiratory or otitis episodes. Several pediatric cohorts have shown clinically relevant associations between asthma control and vitamin D status, whereas a recent Cochrane review reported that people given vitamin D supplements did not have a lower risk of severe asthma attacks. This narrative review integrates mechanistic, observational, and interventional evidence relevant to pediatric wheezing, bronchial asthma, and respiratory tract infections, and discusses practical definitions of deficiency and age-appropriate supplementation. Maintaining adequate vitamin D status may contribute to optimal immune function and may be particularly relevant in children with recurrent respiratory infections, wheezing, or allergic diseases.

KEYWORDS: vitamin D, asthma, wheezing, respiratory infections, children, supplementation

Received: July 15, 2026
Published: September 30, 2026

CORRESPONDENCE

Angela Klain
klainangela95@gmail.com

How to cite this article: Miraglia del Giudice M, Klain A, Dinardo G, et al. The Role of Vitamin D in Pediatric Respiratory Diseases. Italian Journal of Pediatric Allergy and Immunology 2026;40(03):3-10. <https://doi.org/10.53151/2531-3916/2026-2659>

© Copyright by Italian Society of Pediatric Allergy and Immunology



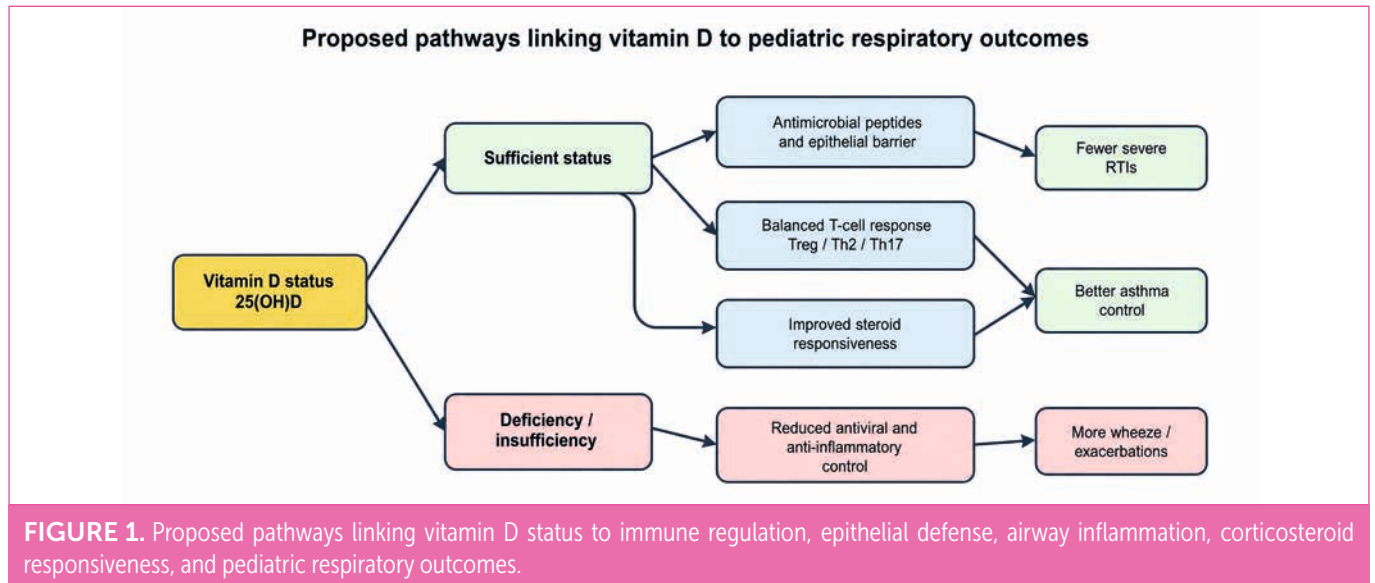
OPEN ACCESS

This is an open access article distributed in accordance with the CC-BY-NC-ND (Creative Commons Attribution-Non-Commercial-NoDerivatives 4.0 International) license. The article can be used by giving appropriate credit and mentioning the license, but only for non-commercial purposes and only in the original version. For further information: <https://creativecommons.org/licenses/by-nc-nd/4.0/deed.en>

INTRODUCTION

Vitamin D has traditionally been considered through the lens of skeletal mineralization, calcium absorption, and prevention of rickets. Its biological role is broader, however, since the vitamin D receptor and vitamin D-metabolizing enzymes are expressed in immune cells, respiratory epithelial cells, and other tissues relevant to host defense. Interest in pediatric respiratory medicine has consequently increased as observational cohorts have linked low circulating 25(OH)D with respiratory infections, recurrent wheeze, asthma morbidity, impaired lung function, and greater treatment burden. A recent pediatric review emphasized that these findings should be interpreted as evidence of a potentially modifiable background factor rather than proof that vitamin D deficiency is an isolated cause of respiratory disease¹.

This distinction is clinically important. Serum 25(OH)D is influenced by sunlight exposure, diet, supplementation, skin phototype, adiposity, season, chronic illness, medication use, and socioeconomic circumstances. In contemporary pediatric populations, reduced outdoor activity and limited sunlight exposure may diminish cutaneous vitamin D synthesis and contribute to the high prevalence of low 25(OH)D concentrations. Several of these determinants are also associated with respiratory outcomes through independent pathways. Obesity, for example, may lower measured vitamin D status through volumetric dilution and is associated with asthma symptoms and altered treatment response. A balanced review must therefore



separate biological plausibility, association, and therapeutic efficacy. Mechanistic studies can identify pathways through which inadequate vitamin D signaling may influence airway defense or inflammation. Cohort studies can establish clinically meaningful patterns and generate hypotheses. Randomized trials are needed to determine whether supplementation changes outcomes, and their interpretation depends on baseline deficiency, dose, dosing interval, adherence, background therapy, and the specific respiratory phenotype under investigation². The conceptual pathways considered in this review are summarized in Figure 1.

1. VITAMIN D DEFICIENCY AS A PRIMARY DRIVER OF AIRWAY INFLAMMATION

The biologically active metabolite 1,25-dihydroxyvitamin D binds to the nuclear vitamin D receptor and modifies transcription in immune and structural cells. Respiratory epithelial cells can participate in local vitamin D metabolism, allowing circulating substrate to influence tissue-level responses. In innate immunity, vitamin D signaling promotes antimicrobial peptides such as cathelicidin and defensins, supports epithelial-barrier integrity, and may enhance the capacity of mucosal surfaces to respond to viral and bacterial challenge. These mechanisms provide a plausible link between profound deficiency and susceptibility to respiratory infection, particularly when other nutritional or environmental vulnerabilities coexist².

Vitamin D also participates in adaptive immune regulation. Experimental models have described its effects on antigen-presenting cells, regulatory T cells, and T-helper-cell polarization.

Adequate signaling tends to limit excessive pro-inflammatory Th1 and Th17 activity and to support regulatory pathways, while the effects on Th2-dominant allergic inflammation are context dependent. This is relevant to asthma because airway disease is not a single immunological entity: preschool viral wheeze, eosinophilic allergic asthma, obesity-associated symptoms, and severe therapy-resistant asthma differ in cellular pathways, natural history, and response to treatment.

The epithelial barrier offers a second point of convergence. Recurrent viral injury, allergen exposure, pollutants, and tobacco smoke can increase epithelial permeability and release alarmins that activate downstream inflammatory cascades. Vitamin D cannot neutralize these exposures, but adequate signaling may contribute to barrier maintenance and antimicrobial readiness. A deficiency state may therefore operate as one component of a broader vulnerability rather than as a sufficient cause of airway inflammation.

Potential interactions with glucocorticoid pathways have generated particular interest. Vitamin D has been associated experimentally with regulatory cytokine responses and molecular processes involved in corticosteroid sensitivity. Airway smooth-muscle proliferation and extracellular-matrix deposition are additional candidate mechanisms linking inadequate signaling to remodeling. These observations are biologically coherent with clinical associations between low 25(OH)D, higher corticosteroid requirements, and severe asthma; nevertheless, mechanistic coherence alone does not establish that supplementation reverses established steroid resistance or airway remodeling in children²⁻⁴.

The threshold at which these non-skeletal pathways becomes clinically relevant is uncertain. Severe deficiency is more likely to compromise multiple physiological systems, whereas small differences within the broadly sufficient range may have little respiratory consequence.-

2. THE INTRICATE LINK BETWEEN VITAMIN D AND PEDIATRIC WHEEZING

Wheezing during the first years of life is a symptom rather than a diagnosis. Episodes may accompany viral bronchiolitis, recurrent infection, early allergic sensitization, anatomical abnormalities, or the evolving phenotype of childhood asthma. Many children wheeze transiently and later become asymptomatic, whereas a smaller subgroup develops persistent disease. Any analysis of vitamin D must therefore consider the timing of measurement and the clinical trajectory rather than treating all wheezing disorders as equivalent.

Prospective birth cohorts have been useful because cord-blood measurements precede most postnatal respiratory events. In a New Zealand cohort of 922 newborns, cord-blood 25(OH)D below 25 nmol/L, compared with 75 nmol/L or higher, was associated with respiratory infection by 3 months of age, with an odds ratio of 2.16 (95% confidence interval 1.35-3.46). Lower concentrations were also associated with wheeze at 15 months, 3 years, and 5 years, but not with incident asthma by age 5⁵.

In the Copenhagen randomized trial, pregnant women received additional vitamin D3 2400 IU/day or placebo from gestational week 24, while all participants also received 400 IU/day as usual care. Persistent wheeze by age 3 occurred in 16% of children in the higher-dose group and 20% in the control group; the difference was not statistically significant, although troublesome lung symptoms were less frequent in offspring exposed to the higher dose⁶.

A second large randomized trial, the Vitamin D Antenatal Asthma Reduction Trial (VDAART), evaluated 4,400 IU/day of vitamin D3 versus 400 IU/day in pregnant women at increased risk of having a child with asthma. At 3 years of age, the incidence of asthma or recurrent wheeze was 6.1% lower in the higher-dose group, although this difference did not reach statistical significance. Notably, a pooled analysis of VDAART and the Copenhagen trial suggested a significant 26% reduction in the risk of asthma or recurrent wheeze by age 3, with a stronger effect among mothers who had adequate vitamin D status early in pregnancy⁷.

Taken together, these findings suggest that vitamin D may have a clinically relevant role in respiratory health during early life, particularly when adequate maternal and infant vitamin D status is achieved during critical windows of immune and lung development. Although individual trials have not always shown statistically significant reductions in persistent wheeze or asthma, the overall direction of effect is consistently favorable, and pooled analyses support a potential preventive benefit. These data strengthen the rationale for preventing vitamin D deficiency during pregnancy and early childhood, especially in populations at increased risk of recurrent wheeze or respiratory infections.

3. BRONCHIAL ASTHMA AND THE VITAMIN D AXIS

Asthma is a heterogeneous chronic respiratory disease characterized by variable symptoms, expiratory airflow limitation, and airway inflammation^{8,9}.

Several pediatric cohorts report clinically relevant associations about asthma control and vitamin D status. In the Childhood Asthma Management Program, serum 25(OH)D was measured in 1024 children with mild-to-moderate persistent asthma. Concentrations of 30 ng/mL or lower were found in 35% of participants. After adjustment for age, sex, body mass index, income, treatment group, and other covariates, insufficient status was associated with higher odds of asthma-related hospitalization or emergency department care over 4 years, with an odds ratio of 1.5 (95% confidence interval 1.1-1.9)¹⁰.

More pronounced gradients have been described in severe asthma. In a cohort comparing children with severe therapy-resistant asthma, children with moderate asthma, and healthy controls, median 25(OH)D concentrations were lowest in the severe group and highest in controls. Lower levels correlated with poorer Asthma Control Test scores, lower lung function, more exacerbations, greater inhaled corticosteroid exposure, and increased airway smooth-muscle mass in the biopsy subgroup¹¹.

These data support testing in selected children when deficiency is plausible, especially in severe or difficult-to-control disease accompanied by obesity, restricted outdoor activity, malabsorption, dietary limitation, darker skin phototype, or chronic systemic therapy. However, a 2023 Cochrane review of 20 randomized trials, with 1155 children and 1070 adults, reported contrasting results. Vitamin D did not reduce the proportion of participants who experienced at least one asthma exacerbation requiring systemic corticosteroids; the pooled odds ratio was 1.04 (95% confidence interval 0.81-1.34). The certainty of this conclusion was strongest for the populations actually enrolled, while children with severe asthma and profound baseline deficiency were underrepresented.

A secondary CAMP analysis provides additional information. Among children treated with inhaled corticosteroids, those who were vitamin D deficient had a smaller increase in pre-bronchodilator FEV1 over 8-12 months than insufficient or sufficient peers: 140 mL compared with 330 mL and 290 mL, respectively¹².

More recent evidence has also suggested a potential benefit of supplementation in selected pediatric populations. A 2024 systematic review and meta-analysis of randomized controlled trials in children found that vitamin D supplementation was associated with a lower overall risk of asthma exacerbations (RR 0.62; 95% CI 0.44-0.87) and with improvement in FEV1. The benefit appeared more evident with standard daily supplementation schedules, supporting the possibility that correction of inadequate vitamin D status may be clinically relevant in selected children with asthma¹³.

A 7-month randomized placebo-controlled trial carried out in 2026 in 236 preschool children with moderate-to-severe virus-induced asthma also supports a potential immunomodulatory

effect of vitamin D. Children received two oral boluses of vitamin D3 100,000 IU, administered 3.5 months apart, plus 400 IU/day for 7 months. Compared with placebo, supplementation was associated with a greater reduction in C-reactive protein (adjusted β -3.154 mg/L; 95% CI, -5.978 to -0.330) and blood neutrophil counts at 7 months (adjusted β -0.657×10^9 cells/L; 95% CI, -1.288 to -0.026). No significant effects were observed for eosinophils, lymphocytes, neutrophil-to-lymphocyte ratio, eosinophil-to-lymphocyte ratio, or total IgE ¹⁴.

However, a recent Cochrane review reported that those given vitamin D supplements did not have a lower risk of severe asthma attacks ¹⁵. The consistent association between low 25(OH)D concentrations and greater asthma morbidity, together with signals of benefit in selected trials and meta-analyses, supports assessment and correction of vitamin D deficiency as part of an individualized approach to children with severe, poorly controlled, or high-risk asthma.

4. BROAD-SPECTRUM IMPLICATIONS FOR OTHER RESPIRATORY TRACT INFECTIONS

Acute respiratory tract infections are among the most frequent causes of pediatric consultation, antimicrobial prescribing, school absence, and hospitalization.¹⁶ Vitamin D is relevant because epithelial defense and antimicrobial peptides are activated early in infection, while seasonal reductions in sunlight exposure coincide with periods of high viral circulation in many regions. Seasonality alone does not prove causation, but it strengthens the rationale for trials focused on deficient populations and regular physiological dosing.

In 2017, an individual-participant-data meta-analysis that included 25 randomized controlled trials identified a modest overall reduction in acute respiratory tract infection risk with vitamin D supplementation. The adjusted odds ratio was 0.88 (95% confidence interval 0.81-0.96). Protection was stronger among participants receiving daily or weekly supplementation without additional bolus doses and among those with baseline 25(OH)D below 25 nmol/L ¹⁷.

The dose-pattern finding is clinically relevant. Large intermittent boluses create a different pharmacokinetic exposure compared to regular daily or weekly intake and may not reproduce physiological availability to immune tissues. The stronger effect in profound deficiency is also consistent with a threshold model in which correction of a limiting substrate is more important than raising concentrations that are already adequate.

Of note, a meta-analysis combines heterogeneous infections, age groups, settings, and dosing regimens. It is thus more useful as evidence that baseline status and dosing schedule matter, and that nutritional correction may have the greatest respiratory relevance in children with very low concentrations.

Disease-specific trials offer concrete pediatric endpoints. In 116 children with recurrent acute otitis media, supplementation with vitamin D 1000 IU/day for four months reduced the number of

children experiencing at least one new episode compared with placebo (26 versus 38; $p=0.03$). The clearest difference was observed for uncomplicated episodes, and protection was greater when serum 25(OH)D reached 30 ng/mL or higher ¹⁸.

In vitamin D-deficient Mongolian schoolchildren, fortified milk providing 300 IU/day increased median 25(OH)D from 7 to 19 ng/mL and reduced parent-reported acute respiratory infections. Mean episode counts were 0.45 in the vitamin D group and 0.80 in controls, corresponding to a rate ratio of 0.52.¹⁹ The magnitude of this result should be interpreted in the context of profound baseline deficiency, in which a relatively modest dose corrected an important nutritional gap.

A randomized trial in schoolchildren in Japan evaluated vitamin D3 1200 IU/day during winter. Influenza A infection occurred in 10.8% of supplemented children and 18.6% of those receiving placebo, with a relative risk of 0.58 (95% confidence interval 0.34-0.99). Fewer asthma attacks were reported in the subgroup with asthma ²⁰.

Overall, the available evidence supports a clinically meaningful role of vitamin D in the prevention of acute respiratory tract infections, particularly in children with low baseline 25(OH)D concentrations and when supplementation is provided regularly at physiological daily or weekly doses. The consistency of findings across heterogeneous settings, including recurrent otitis media, winter influenza, and populations with profound deficiency, suggests that correcting inadequate vitamin D status may strengthen mucosal defence and reduce infection burden.

5. CLINICAL MANAGEMENT AND SUPPLEMENTATION

Clinical management should distinguish nutritional prevention, treatment of documented deficiency, and experimental use of vitamin D as a respiratory therapy. Serum total 25(OH)D is the preferred marker of vitamin D status. Routine population screening is not required, but assessment is reasonable when meaningful risk factors or clinical features are present. Relevant factors include obesity, malabsorption, restricted diets, limited sunlight exposure, darker skin phototype, chronic kidney or liver disease, recurrent fractures or bone pain, and treatment with anticonvulsants, glucocorticoids, or other drugs that alter vitamin D metabolism ^{21,22}.

Interpretation begins with the assay and clinical context. Total serum 25(OH)D reflects cutaneous synthesis and dietary intake better than circulating 1,25-dihydroxyvitamin D, which is tightly regulated and may remain normal or increase during deficiency. Values should be considered alongside age, season, growth, calcium intake, comorbidities, and the reason for testing. A single borderline result in an otherwise healthy child should not be treated in the same way as profound deficiency in a child with malabsorption.

Operational cut-offs differ among organizations. The Italian pediatric consensus defines severe deficiency as a serum 25(OH)D concentration below 10 ng/mL (25 nmol/L), deficiency as below 20

TABLE I. Key pediatric clinical evidence linking vitamin D status or supplementation with respiratory outcomes.

Study	Design and population	Vitamin D assessment/intervention	Respiratory outcome	Main finding
Camargo et al.	Prospective birth cohort; 922 newborns	Cord-blood 25(OH)D: <25 versus $\geq$ 75 nmol/L	Early respiratory infection; wheeze and asthma to age 5 years	Low cord-blood 25(OH)D was associated with respiratory infection by 3 months (OR 2.16; 95% CI 1.35-3.46) and wheeze during early childhood, but not with incident asthma by age 5 years
Chawes et al.	Randomized antenatal supplementation trial	Additional vitamin D3 2400 IU/day from gestational week 24; all mothers received 400 IU/day as usual care	Persistent wheeze and troublesome lung symptoms by age 3 years	Persistent wheeze was numerically lower but not significantly reduced (16% vs. 20%); troublesome lung symptoms were less frequent in the higher-dose group
Litonjua et al. (VDAART)	Randomized antenatal supplementation trial in pregnant women at increased asthma risk	Vitamin D3 4400 IU/day versus 400 IU/day	Asthma or recurrent wheeze by age 3 years	Asthma or recurrent wheeze was 6.1% less frequent in the higher-dose group, although the difference was not statistically significant
Brehm et al.	CAMP cohort; 1024 children with mild-to-moderate persistent asthma	Baseline serum 25(OH)D	Asthma-related hospitalization or emergency department care over 4 years	25(OH)D $\leq$ 30 ng/mL was present in 35% of participants and was associated with increased risk of severe asthma-related events (OR 1.5; 95% CI 1.1-1.9)
Gupta et al.	Cohort of children with severe therapy-resistant asthma, moderate asthma, and healthy controls	Serum 25(OH)D	Asthma control, lung function, exacerbations, corticosteroid exposure, and airway remodeling	Lower concentrations correlated with poorer control, lower lung function, more exacerbations, greater inhaled corticosteroid exposure, and increased airway smooth-muscle mass
Williamson et al.	Cochrane systematic review; 20 randomized asthma trials	Vitamin D supplementation	Exacerbations requiring systemic corticosteroids	No reduction in the proportion of participants with at least one treated exacerbation (OR 1.04; 95% CI 0.81-1.34); children with severe asthma and profound deficiency were underrepresented
Wu et al.	Secondary CAMP analysis in children receiving inhaled corticosteroids	Vitamin D status during follow-up	Change in pre-bronchodilator FEV1	Vitamin D-deficient children had a smaller FEV1 gain over 8-12 months than insufficient or sufficient peers (approximately 140 vs 330 and 290 mL).
Fedora et al.	Systematic review and meta-analysis of pediatric randomized controlled trials	Vitamin D supplementation	Asthma exacerbations and FEV1	Supplementation was associated with fewer exacerbations overall (RR 0.62; 95% CI 0.44-0.87) and improved FEV1; the signal appeared stronger with regular daily regimens
Martineau et al.	Individual participant data meta-analysis; 25 randomized controlled trials	Daily, weekly, or bolus vitamin D supplementation	Acute respiratory tract infections	Supplementation modestly reduced acute respiratory tract infection risk overall (adjusted OR 0.88; 95% CI 0.81-0.96), particularly with daily/weekly dosing and baseline 25(OH)D <25 nmol/L.

(continues)

TABLE I. (follows). Key pediatric clinical evidence linking vitamin D status or supplementation with respiratory outcomes.

Study	Design and population	Vitamin D assessment/ intervention	Respiratory outcome	Main finding
Marchisio et al.	Randomized placebo-controlled trial; 116 otitis-prone children	Vitamin D 1000 IU/day for 4 months	Acute otitis media recurrence	Fewer children experienced at least one new episode than with placebo (26 vs. 38; $p=0.03$)
Camargo et al.	Randomized trial in vitamin D-deficient Mongolian schoolchildren	Fortified milk providing vitamin D3 300 IU/day	Acute respiratory infections	Median 25(OH)D increased from 7 to 19 ng/mL; acute respiratory infections were less frequent (rate ratio 0.52)
Urashima et al.	Randomized placebo-controlled winter trial in Japanese schoolchildren	Vitamin D3 1200 IU/day	Influenza A; asthma attacks in asthma subgroup	Influenza A occurred less frequently with vitamin D than placebo (10.8% vs 18.6%; RR 0.58; 95% CI 0.34-0.99); fewer asthma attacks were reported in the asthma subgroup
El Abd et al.	Nested study within a 7-month randomized placebo-controlled trial; 236 preschool children aged 1-5 years with moderate-to-severe virus-induced asthma	Vitamin D3 100,000 IU orally at baseline and 3.5 months, plus 400 IU/day for 7 months, versus placebo	Systemic inflammatory biomarkers: CRP and blood-cell counts	Compared with placebo, vitamin D supplementation significantly reduced CRP over follow-up (adjusted β -3.154 mg/L) and blood neutrophils at 7 months (adjusted β -0.657 $\times 10^9$ cells/L), with no significant effect on eosinophils, lymphocytes, NLR, ELR, or total IgE

CAMP: Childhood Asthma Management Program; FEV1: forced expiratory volume in 1 second; OR: odds ratio; RR: relative risk; VDAART: Vitamin D Antenatal Asthma Reduction Trial; 25(OH)D: 25-hydroxyvitamin D.

ng/mL (50 nmol/L), insufficiency as 20-29 ng/mL (50-74 nmol/L), and sufficiency as 30 ng/mL (75 nmol/L) or higher²¹. These categories are useful for clinical communication but should not be interpreted as universal biological boundaries.

The 2024 Endocrine Society guideline did not identify a serum 25(OH)D target that reliably predicts disease-prevention benefit across populations²². This position reflects uncertainty about non-skeletal thresholds and cautions against using increasingly high concentrations as a therapeutic goal. It does not negate the need to prevent rickets or correct clear deficiency.

For routine prevention, vitamin D3 400 IU/day is recommended for term infants from the first days of life through 12 months, irrespective of feeding mode; infants with risk factors may require up to 1000 IU/day. From 1 to 18 years, 600 IU/day is the usual reference intake, with up to 1000 IU/day considered when reduced sunlight exposure or multiple risk factors are present.

Preventive intake should account for all sources, including formula, fortified foods, multivitamins, and prescribed preparations. Clear communication about product concentration is essential because drops may contain very different amounts per dose. Families should be advised to use an age-appropriate product and a calibrated dispenser and to avoid combining several vitamin D-containing supplements without reviewing the total daily intake.

In children and adolescents aged 1-18 years with confirmed asymptomatic vitamin D deficiency [25(OH)D <20 ng/mL], 2,000 IU/day for 6-8 weeks (8 weeks in adolescents) may be used. Serum 25(OH)D should subsequently be reassessed and age-appropriate

maintenance initiated¹⁴. Higher or prolonged regimens require specialist supervision in obesity, malabsorption, chronic kidney or liver disease, granulomatous disorders, or during treatment with drugs that accelerate vitamin D catabolism. Monitoring may include 25(OH)D, calcium, phosphate, alkaline phosphatase, parathyroid hormone, renal function, and urinary calcium according to the clinical situation. The purpose is to document correction while avoiding hypercalcemia, hypercalciuria, nephrocalcinosis, or unnecessary accumulation.

Table I summarizes the pediatric studies most useful for clinical interpretation. Table II reports operational definitions and a practical dosing framework.

The relationship between obesity, vitamin D, and asthma illustrates the need for integrated care. Adiposity can lower circulating 25(OH)D and is associated with dyspnea, altered mechanics, systemic inflammation, and reduced responsiveness to some asthma treatments. Correcting deficiency may be appropriate, but the relationship between obesity and asthma probably implies different endotypes and different inflammatory mechanisms in which vitamin D is only one of the factors.

Childhood obesity is an increasingly important public-health problem and a major contributor to respiratory morbidity. The relationship between obesity, vitamin D, and asthma therefore illustrates the need for integrated care. Adiposity can lower circulating 25(OH)D through volumetric dilution, sequestration within adipose tissue, reduced outdoor activity, and dietary patterns associated with lower vitamin D intake. Children and adolescents with obesity may consequently be at

TABLE II. Operational definitions of vitamin D status and age-appropriate supplementation in pediatric practice.

Clinical context	25(OH)D / age	Suggested approach
Severe deficiency	<10 ng/mL (<25 nmol/L)	Clinical assessment and correction under medical supervision
Deficiency	<20 ng/mL (<50 nmol/L)	Correct documented deficiency and reassess
Insufficiency	20-29 ng/mL (50-74 nmol/L)	Interpret with age, intake, season, and risk factors
Sufficiency	≥30 ng/mL (≥75 nmol/L)	Maintain age-appropriate intake
Routine prevention	Birth to 12 months	Vitamin D3 400 IU/day
Routine prevention	1-18 years	Vitamin D3 600 IU/day
Asymptomatic deficiency	Pediatric age	2000 IU/day or 50,000 IU once weekly for 6-8 weeks

Doses are a general framework and do not replace individual clinical assessment. Higher or prolonged regimens require specialist supervision.

increased risk of vitamin D insufficiency or deficiency and may require closer biochemical follow-up and individualized supplementation strategies when deficiency is confirmed.

At the same time, obesity is independently associated with dyspnea, reduced functional residual capacity, altered chest-wall and airway mechanics and systemic low-grade inflammation.

Overall, maintaining adequate vitamin D status represents a simple, safe, and potentially valuable component of comprehensive care in children with respiratory symptoms. Early recognition and correction of deficiency may support musculoskeletal health, immune function, and overall respiratory well-being within an individualized multidisciplinary approach.

CONCLUSION

Italian children show a clear seasonal variation in vitamin D status, with lower serum 25(OH)D concentrations particularly during winter and spring, when reduced sunlight exposure limits cutaneous vitamin D synthesis²³. It is known that Vitamin D deficiency is best understood as a modifiable vulnerability that may interact with epithelial defense, infection susceptibility, allergic inflammation, asthma severity, and treatment response rather than as a single cause of pediatric respiratory disease^{1,2}.

The strongest observational signals are phenotype specific: very low cord-blood 25(OH)D is associated with early respiratory infection and wheeze; pediatric asthma cohorts associate lower concentrations with severe events, poorer control, greater treatment burden, and weaker lung-function response. Selected infection trials and meta-analytic data indicate a potential benefit when the deficiency is profound, dosing is regular, or the population has a high burden of respiratory infection¹⁰⁻¹³.

Vitamin D should be regarded as a relevant nutritional and immunological factor in pediatric respiratory health, rather than as a stand-alone treatment for asthma or recurrent respiratory infections. Its biological effects on epithelial integrity, antimicrobial peptide production, immune regulation, and glucocorticoid-related pathways

provide a plausible framework for the associations observed across different respiratory phenotypes. However, the clinical relevance of vitamin D status is likely to depend on the underlying condition, baseline 25(OH)D concentration, age, season, comorbidities, and pattern of supplementation.

Taken together, the pediatric studies reviewed do not support vitamin D supplementation as a universal intervention for acute respiratory infections in children. In asthma, any potential benefit may be confined to selected subgroups, including children with profound baseline vitamin D deficiency [25(OH)D <10 ng/mL], severe steroid-resistant asthma, or obesity. Clinical efficacy in these subgroups remains to be established, and correction of deficiency should form part of individualized care rather than replace established asthma treatment. For wheezing, the available evidence highlights the importance of adequate maternal and infant vitamin D status during critical windows of immune and lung development. Further studies are needed to define optimal dosing strategies and clarify the role of vitamin D supplementation in other pediatric respiratory conditions.

Ethical consideration

Not applicable. This is a narrative review of published literature and does not involve new studies with human participants, animals, or identifiable patient data.

Funding

No funding.

Conflicts of interest statement

No conflict of interest to declare.

Authors' contribution

Conceptualization: A.K. and M.M.d.G. Methodology: A.K. and M.M.d.G. Investigation: A.K. Writing-Original Draft: A.K. Writing-Review & Editing: S.C., G.D., C.G., N.M., C.I., M.M.d.G. Supervision: C.I. and M.M.d.G. Project Administration: M.M.d.G.

References

- ¹ Indolfi C, Klain A, Dinardo G, et al. Mini-review on vitamin D in pediatric population and its role in respiratory and atopic disorders. *Mini Rev Med Chem* 2024;24:1386-1394. <https://doi.org/10.2174/0113895575284873240212045431>.
- ² Miraglia Del Giudice M, Indolfi C, Strisciuglio C. Vitamin D: immunomodulatory aspects. *J Clin Gastroenterol* 2018;52(Suppl 1):S86-S88. <https://doi.org/10.1097/MCG.0000000000001112>
- ³ Miraglia Del Giudice M, Allegorico A. The role of vitamin D in allergic diseases in children. *J Clin Gastroenterol* 2016;50(suppl 2):S133-S135. <https://doi.org/10.1097/MCG.0000000000000679>
- ⁴ Miraglia Del Giudice M, Maiello N, Allegorico A, et al. Lactobacillus reuteri DSM 17938 plus vitamin D3 as ancillary treatment in allergic children with asthma. *Ann Allergy Asthma Immunol* 2016;117:710-712. <https://doi.org/10.1016/j.anai.2016.09.004>
- ⁵ Camargo CA Jr, Ingham T, Wickens K, et al. Cord-blood 25-hydroxyvitamin D levels and risk of respiratory infection, wheezing, and asthma. *Pediatrics* 2011;127:e180-187. <https://doi.org/10.1542/peds.2010-0442>.
- ⁶ Chawes BL, Bønnelykke K, Stokholm J, et al. Effect of vitamin D3 supplementation during pregnancy on risk of persistent wheeze in the offspring: a randomized clinical trial. *JAMA* 2016;315:353-361. <https://doi.org/10.1001/jama.2015.18318>.
- ⁷ Litonjua AA, Carey VJ, Laranjo N, et al. Effect of Prenatal Supplementation With Vitamin D on Asthma or Recurrent Wheezing in Offspring by Age 3 Years: The VDAART Randomized Clinical Trial. *JAMA* 2016;315:362-370. <https://doi.org/10.1001/jama.2015.18589>.
- ⁸ Global Initiative for Asthma. Global strategy for asthma management and prevention. 2026. Available at: <https://ginasthma.org/2026-gina-strategy-report/>. Accessed June 18, 2026.
- ⁹ Miraglia Del Giudice M, Allegorico A, Grandone A, et al. Vitamin D and bronchial inflammation in asthmatic children. *J Biol Regul Homeost Agents* 2015;29(2 Suppl 1):125-129.
- ¹⁰ Brehm JM, Schuemann B, Fuhlbrigge AL, et al. Serum vitamin D levels and severe asthma exacerbations in the Childhood Asthma Management Program study. *J Allergy Clin Immunol* 2010;126:52-58.e5. <https://doi.org/10.1016/j.jaci.2010.03.043>
- ¹¹ Gupta A, Sjoukes A, Richards D, et al. Relationship between serum vitamin D, disease severity, and airway remodeling in children with asthma. *Am J Respir Crit Care Med* 2011;184:1342-1349. <https://doi.org/10.1164/rccm.201107-1239OC>.
- ¹² Wu AC, Tantisira K, Li L, et al. Effect of vitamin D and inhaled corticosteroid treatment on lung function in children. *Am J Respir Crit Care Med* 2012;186:508-513. <https://doi.org/10.1164/rccm.201202-0351OC>.
- ¹³ Fedora K, Setyoningrum RA, Aina Q, et al. Vitamin D supplementation decrease asthma exacerbations in children: a systematic review and meta-analysis of randomized controlled trials. *Ann Med* 2024;56:2400313. <https://doi.org/10.1080/07853890.2024.2400313>.
- ¹⁴ El Abd A, Trottier H, Tse SM, et al. Impact of vitamin D status and supplementation on exploratory inflammatory biomarkers in preschoolers with asthma. *Ann Allergy Asthma Immunol* 2026;137:53-63. <https://doi.org/10.1016/j.anai.2026.03.022>.
- ¹⁵ Williamson A, Martineau AR, Sheikh A, et al. Vitamin D for the management of asthma. *Cochrane Database Syst Rev* 2023;2:CD011511. <https://doi.org/10.1002/14651858.CD011511.pub3>.
- ¹⁶ Miraglia Del Giudice M, Indolfi C, Dinardo G, et al. Vitamin D status can affect COVID-19 outcomes also in pediatric population. *PharmaNutrition* 2022;22:100319. <https://doi.org/10.1016/j.phanu.2022.100319>.
- ¹⁷ Martineau AR, Jolliffe DA, Hooper RL, et al. Vitamin D supplementation to prevent acute respiratory tract infections: systematic review and meta-analysis of individual participant data. *BMJ* 2017;356:i6583. <https://doi.org/10.1136/bmj.i6583>.
- ¹⁸ Marchisio P, Consonni D, Baggi E, et al. Vitamin D supplementation reduces the risk of acute otitis media in otitis-prone children. *Pediatr Infect Dis J* 2013;32:1055-1060. <https://doi.org/10.1097/INF.0b013e31829be0b0>.
- ¹⁹ Camargo CA Jr, Ganmaa D, Frazier AL, et al. Randomized trial of vitamin D supplementation and risk of acute respiratory infection in Mongolia. *Pediatrics* 2012;130:e561-567. <https://doi.org/10.1542/peds.2011-3029>.
- ²⁰ Urashima M, Segawa T, Okazaki M, et al. Randomized trial of vitamin D supplementation to prevent seasonal influenza A in schoolchildren. *Am J Clin Nutr* 2010;91:1255-1260. <https://doi.org/10.3945/ajcn.2009.29094>.
- ²¹ Saggese G, Vierucci F, Prodam F, et al. Vitamin D in pediatric age: consensus of the Italian Pediatric Society and the Italian Society of Preventive and Social Pediatrics, jointly with the Italian Federation of Pediatricians. *Ital J Pediatr* 2018;44:51. <https://doi.org/10.1186/s13052-018-0488-7>.
- ²² Demay MB, Pittas AG, Bikle DD, et al. Vitamin D for the prevention of disease: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab* 2024;109:1907-1947. <https://doi.org/10.1210/clinem/dgae290>.
- ²³ Galeazzi T, Quattrini S, Pjetraj D, et al. Vitamin D status in healthy Italian school-age children: a single-center cross-sectional study. *Ital J Pediatr* 2023;49:27. <https://doi.org/10.1186/s13052-023-01422-x>.