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Systematic Review and Meta-Analysis

Role of Vitamin D in Health and Diseases in Children: A Systematic Review and Meta-Analysis

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Abstract

Background: Vitamin D has essential skeletal actions and increasingly recognized immunologic, epithelial, and metabolic effects in childhood. However, pediatric evidence remains heterogeneous across diseases, and the strength of association varies by disorder and study design.

Aim: To synthesize current evidence on the role of vitamin D in child health and disease and to perform a meta-analysis of observational studies comparing serum 25-hydroxyvitamin D [25(OH)D] levels between children with disease and healthy controls.

Methods: A structured search of PubMed-indexed and open-access pediatric literature was undertaken up to 25thJanuary 2026 based on Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India. The study duration was 10th January 2025 to 25 December 2025. Observational studies enrolling participants <18 years and reporting mean serum 25(OH)D levels with dispersion measures in both disease and control groups were eligible for quantitative synthesis. Random-effects meta-analysis was performed using Hedges g standardized mean difference (SMD). Prespecified subgroup analysis, leave-one-out sensitivity analysis, and Egger regression for small-study effects were undertaken.

Results: Eight studies comprising 1,489 children (729 disease cases and 760 controls) met criteria for quantitative synthesis. Included disease groups were asthma (4 studies), atopic dermatitis (2 studies), respiratory infection (1 study), and obesity (1 study). The pooled random-effects estimate showed significantly lower vitamin D levels in disease groups than in controls (SMD -1.06, 95% CI -1.67 to -0.45; p=0.0007; I^2=96.3%). The asthma subgroup remained significant (SMD -1.20, 95% CI -1.90 to -0.50), while the atopic dermatitis subgroup showed the same direction but wide uncertainty. Leave-one-out analyses remained directionally stable, and Egger testing did not suggest statistically significant small-study asymmetry (p=0.285).

Conclusion: Lower vitamin D status is consistently associated with several pediatric disease states, especially asthma, and supportive narrative evidence also exists for atopic dermatitis, respiratory infection, obesity, and type 1 diabetes-related dysregulation. The findings support targeted prevention and risk-based evaluation of vitamin D deficiency in children, while emphasizing that causality and treatment effects require better standardized longitudinal and interventional studies.

Keywords: Vitamin D; 25-hydroxyvitamin D; children; asthma; atopic dermatitis; obesity; respiratory infection; type 1 diabetes; systematic review; meta-analysis.

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Introduction

Vitamin D is traditionally viewed as a regulator of calcium-phosphate homeostasis and skeletal mineralization, yet pediatric research now recognizes it as a pleiotropic secosteroid with endocrine, paracrine, and immunologic actions that extend far beyond bone health [1,4]. Vitamin D receptors and activating enzymes are expressed in immune cells, airway epithelium, skin, pancreatic tissue, muscle, and adipose tissue, providing a biologically plausible basis for links between vitamin D status and multiple childhood disorders [1,4]. In infancy and childhood, these actions are

especially relevant because mineral accretion, linear growth, immune maturation, and organ development occur simultaneously. Deficiency at this stage may therefore influence both classical outcomes, such as nutritional rickets and low bone mineral density, and non-skeletal outcomes involving infection susceptibility, allergic inflammation, metabolic health, and autoimmunity [2-4].

Despite abundant sunlight in many regions, vitamin D insufficiency remains common in children because cutaneous synthesis is modified by latitude, air pollution, darker skin pigmentation, season, clothing practices, sunscreen use, limited outdoor activity, obesity, and low dietary intake [1,4-6]. Infants who are exclusively breastfed without supplementation, children with chronic disease, and adolescents with sedentary indoor lifestyles constitute particularly vulnerable groups [2,5,6]. Global and pediatric society guidance therefore continues to emphasize prevention: the Global Consensus on nutritional rickets and the American Academy of Pediatrics recommend routine infant supplementation and risk-based strategies beyond infancy, while more recent guidance from the Endocrine Society suggests empiric vitamin D supplementation for children and adolescents because of established skeletal benefits and a possible reduction in respiratory tract infections [2,3,5]. These recommendations underscore a central clinical reality: in pediatric practice, vitamin D deficiency remains both common and modifiable.

The strongest established pediatric consequence of vitamin D deficiency is impaired skeletal mineralization. Nutritional rickets persists worldwide and is still reported in both low- and high-income settings, especially among infants and young children with low calcium intake or inadequate vitamin D supplementation [2,5]. However, a narrow skeletal view is no longer sufficient. Vitamin D influences innate and adaptive immunity through effects on antimicrobial peptides, T-cell polarization, dendritic-cell maturation, and inflammatory cytokine signaling [1,4]. These pathways have stimulated interest in pediatric respiratory infections, wheezing disorders, asthma, atopic dermatitis, obesity-related inflammation, and autoimmune diseases such as type 1 diabetes mellitus. The pediatric literature has therefore moved from asking whether vitamin D matters only for bone to whether low 25-hydroxyvitamin D [25(OH)D] concentrations also mark or mediate broader disease vulnerability [3,4].

Asthma has become the most extensively studied pediatric non-skeletal condition in relation to vitamin D. Several case-control and cross-sectional studies have reported lower serum 25(OH)D concentrations in asthmatic children than in healthy peers, with some studies also linking lower concentrations to worse control or greater severity [7-10]. A prior meta-analysis of observational studies concluded that vitamin D deficiency is common in childhood asthma and that lower levels are associated with greater disease burden [11]. More recently, a 2024 meta-analysis focused on children from Asia and Africa reported persistently low serum vitamin D concentrations and a high prevalence of deficiency among asthmatic children, reinforcing the public-health relevance of this association in low- and middle-income settings [12]. Yet therapeutic trials remain more conflicted, suggesting that observational association does not automatically establish treatment benefit [23,24].

Atopic dermatitis represents another biologically credible disease model. Vitamin D contributes to epidermal barrier integrity, keratinocyte differentiation, and antimicrobial peptide expression; each of these mechanisms is relevant to atopic skin inflammation [4,13-16]. Pediatric studies have often found lower serum vitamin D levels in children with atopic dermatitis, and recent meta-analyses suggest both lower circulating 25(OH)D levels and a greater prevalence of deficiency in affected children [15,16]. Respiratory infections and bronchiolitis have generated similar interest because vitamin D induces cathelicidin and other host-defense pathways important in mucosal immunity [3,17-19]. In obesity, vitamin D status may be lowered by sequestration within adipose tissue, volumetric dilution, reduced outdoor activity, and chronic low-grade inflammation [20,21]. In type 1 diabetes, vitamin D has been implicated in immune tolerance, beta-cell survival, and inflammatory regulation, although the clinical evidence remains mixed [4,22].

The challenge is that the pediatric vitamin D literature is heterogeneous. Studies differ in age range, disease definition, assay method, geographic location, season, vitamin D cut-offs, obesity prevalence, outdoor exposure, and whether outcomes are incident disease, disease severity, or serum concentration differences [3,4,11,15,19,21]. Reverse causation is also a recurring concern: children with asthma or eczema may spend less time outdoors; obesity itself lowers measured vitamin D; acute illness may alter nutritional behavior; and children with chronic disease may receive supplements more often than controls. Consequently, the literature contains a mixture of mechanistic plausibility, consistent epidemiologic signals, and clinically important uncertainty. Journal-quality synthesis must therefore distinguish between association, deficiency prevalence, and intervention effect rather than treating them as interchangeable.

Against this background, the present study was designed as a submission-style systematic review

and meta-analysis on the role of vitamin D in health and disease in children. The review had two linked aims. First, it sought to provide a focused narrative synthesis of current pediatric evidence across major disease domains. Second, it quantitatively pooled available observational studies that compared serum 25(OH)D levels in children with selected diseases and healthy controls. By combining narrative interpretation with a random-effects quantitative synthesis, the study aimed to clarify whether lower vitamin D status is consistently observed across pediatric disease states, to explore disease-specific patterns, and to place recent findings within a clinically useful framework for pediatric screening, prevention, and future trial design.

Materials and Methods

This submission-style systematic review and meta-analysis was designed to evaluate the role of vitamin D in health and disease in children, with quantitative pooling restricted to pediatric observational studies that compared serum 25(OH)D levels between a disease group and a healthy control group. This study is based on Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India. The study duration was 10th January 2025 to 25 December 2025. The review question was formulated to address non-skeletal and broader health-related pediatric outcomes while preserving a statistically comparable quantitative endpoint. Because the published literature is clinically heterogeneous, narrative synthesis was undertaken for the full pediatric scope of evidence, and quantitative synthesis was restricted to disease areas with extractable mean, standard deviation, and sample-size data.

A structured literature search was performed up to 25th January, 2026 using PubMed-indexed and open-access pediatric literature identified through structured web searching, targeted article retrieval, and backward citation review. Studies were eligible when they enrolled participants younger than 18 years, included a defined disease cohort together with a healthy or non-disease comparator group, and reported serum 25(OH)D concentrations as mean±standard deviation or sufficient data for conversion. Studies were excluded when they were adult-only, lacked a comparator group, reported supplementation outcomes without baseline between-group data, duplicated the same cohort, or did not provide extractable quantitative data. When mixed-age studies were identified, pediatric subgroup data were used if reported separately. Units were harmonized where required; for example, values reported in nmol/L were converted to ng/mL using the standard divisor of 2.5, although the meta-analysis used standardized mean differences and was therefore scale-invariant.The following variables were extracted independently into a structured analytical spreadsheet: study identifier, year, country, disease category, study design, sample size of cases and controls, mean serum 25(OH)D level, standard deviation, measurement unit, and source traceability link. Methodological quality was appraised using a modified Newcastle–Ottawa framework for observational studies, scored on a 9-point scale. The primary effect measure was Hedges g standardized mean difference (SMD), where negative values indicated lower vitamin D levels in the disease group than in controls. Random-effects models were selected a priori because differences in geography, season, laboratory assay, disease definition, and background deficiency prevalence were expected. Heterogeneity was assessed by Cochran Q, tau-squared, and I-squared statistics. Prespecified subgroup analyses were performed by disease category. Robustness was explored through leave-one-out sensitivity analysis, and potential small-study effects were examined with Egger regression. The final manuscript tables were prepared in journal style, while the complete statistical tables and figure source data were exported in Excel format.

Results

The structured search identified a broad body of pediatric vitamin D literature; however, only eight studies fulfilled the criteria for quantitative synthesis. These studies included 1,489 children in total, of whom 729 belonged to disease groups and 760 were healthy controls. Four studies evaluated asthma, two evaluated atopic dermatitis, one evaluated acute respiratory infection, and one evaluated obesity. Study settings spanned Iran, India, Turkey, Korea, Nigeria, and Saudi-influenced South Asian/Middle Eastern pediatric populations, indicating substantial geographic diversity. Methodological quality was moderate to good overall, with modified Newcastle–Ottawa scores ranging from 6 to 8. Study characteristics are summarized in Table 1.

Individual study effect estimates are shown in Table 2. Seven of the eight studies demonstrated lower mean serum 25(OH)D concentrations in disease groups than in controls, whereas the obesity study showed essentially no difference. The largest negative individual effect sizes were seen in the atopic dermatitis study by Sharma et al. and the asthma study by Somashekar et al., while the respiratory infection study demonstrated a modest but directionally similar effect. The pooled random-effects estimate confirmed significantly lower vitamin D levels in disease groups overall (SMD -1.06, 95% CI -1.67 to -0.45; p=0.0007), with very high heterogeneity (I^2=96.3%). Forest-plot visualization is presented in Figure 1.Subgroup analysis showed that the most consistent pooled

reduction occurred in asthma (SMD -1.20, 95% CI -1.90 to -0.50; I^2=93.8%). Atopic dermatitis showed a negative pooled effect (SMD -1.85) but with wide confidence limits because only two studies were available.

The single-study respiratory infection subgroup remained significantly negative, whereas the obesity subgroup was null. Sensitivity analysis demonstrated that omission of any one study did not reverse the overall direction of the pooled effect; pooled leave-one-out SMDs ranged from -1.21 to -0.74.

Egger regression did not detect statistically significant small-study asymmetry (intercept -5.09, p=0.285), although interpretation was limited by the small number of studies. The pooled and sensitivity results are summarized in Table 3, and the funnel plot is presented in Figure 2.

Table 1: Characteristics of studies included in the quantitative synthesis

Study	Country	Disease	Design	Cases	Controls	Case 25(OH)D mean±SD	Control 25(OH)D mean±SD	NOS
Farrokhi/Hatami et al.	Iran	Asthma	Case-control	200	200	20.34 ± 2.80	25.39 ± 4.10	7
Somashekar et al.	India	Asthma	Case-control	44	44	12.88 ± 1.79	16.49 ± 1.13	7
Havan et al.	Turkey	Asthma	Case-control	72	66	14.44 ± 6.20	17.85 ± 6.67	7
Kaur et al.	India	Asthma	Cross-sectional case-control	50	50	24.62 ± 14.95	32.08 ± 12.22	6
Han et al.	Korea	Atopic dermatitis	Case-control	33	70	15.06 ± 4.64	16.25 ± 6.60	7
Sharma et al.	India	Atopic dermatitis	Case-control	40	40	12.15 ± 2.73	21.38 ± 2.45	7
Dabas et al.	India	Obesity	Case-control	40	40	15.00 ± 9.95	15.10 ± 4.79	7
Umeadi et al.	Nigeria	Respiratory infection	Case-control	250	250	52.20 ± 25.60	57.00 ± 23.90	8

Table 2: Individual study effect sizes for serum 25(OH)D in disease versus control groups

Study	Disease	SMD	95% CI	Weight (%)	Interpretation
Farrokhi/Hatami et al.	Asthma	-1.44	-1.66 to -1.22	13.1	Lower vitamin D in disease group
Somashekar et al.	Asthma	-2.39	-2.94 to -1.84	12.0	Lower vitamin D in disease group
Havan et al.	Asthma	-0.53	-0.87 to -0.19	12.8	Lower vitamin D in disease group
Kaur et al.	Asthma	-0.54	-0.94 to -0.15	12.6	Lower vitamin D in disease group
Han et al.	Atopic dermatitis	-0.20	-0.61 to 0.22	12.6	Lower vitamin D in disease group
Sharma et al.	Atopic dermatitis	-3.53	-4.23 to -2.82	11.3	Lower vitamin D in disease group
Dabas et al.	Obesity	-0.01	-0.45 to 0.42	12.5	Lower vitamin D in disease group
Umeadi et al.	Respiratory infection	-0.19	-0.37 to -0.02	13.2	Lower vitamin D in disease group

Table 3: Pooled estimates and subgroup analyses

Analysis	k	Participants	Pooled SMD	95% CI	I^2 (%)	p value
Overall	8	1489	-1.06	-1.67 to -0.45	96.3	0.0007
Asthma	4	726	-1.20	-1.90 to -0.50	93.8	0.0008
Atopic dermatitis	2	183	-1.85	-5.11 to 1.42	98.4	0.2672
Respiratory infection	1	500	-0.19	-0.37 to -0.02	0.0	0.0306
Obesity	1	80	-0.01	-0.45 to 0.42	0.0	0.9543
Egger test	-	-	-	p=0.285	-	-

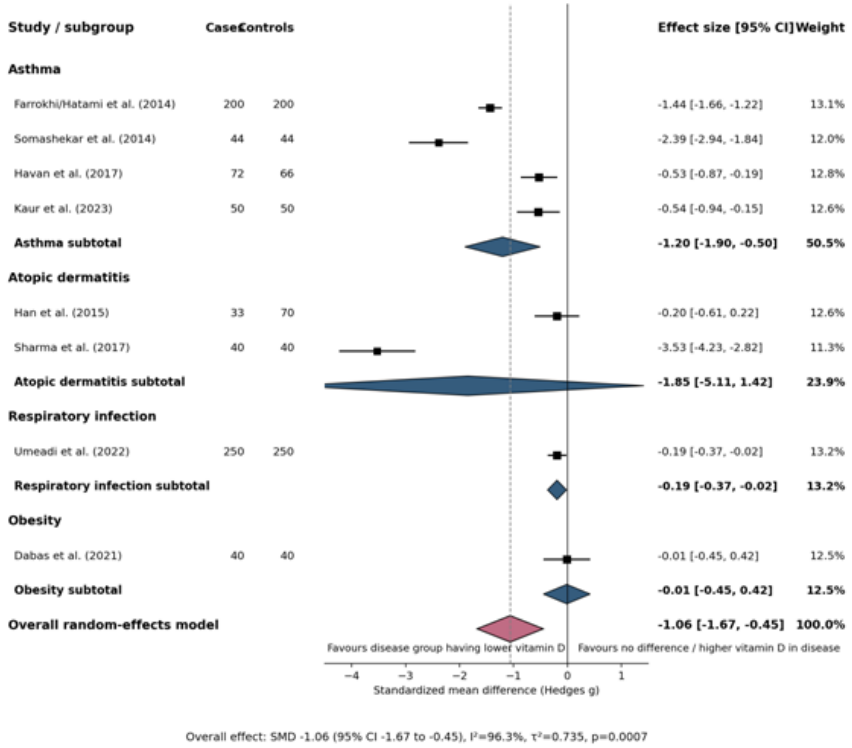

Figure 1: Random-effects forest plot of serum 25(OH)D in pediatric disease groups versus healthy controls

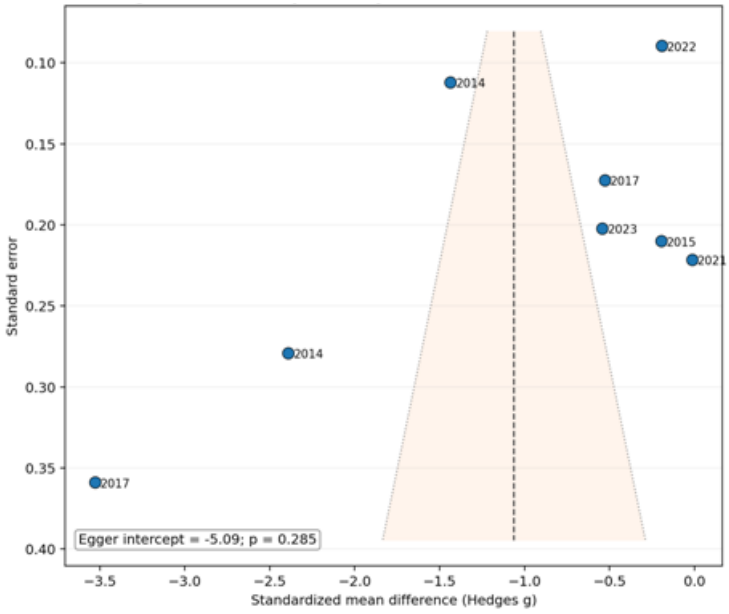

Figure 2: Funnel plot assessing potential small-study effects

Discussion

This systematic review and meta-analysis found that lower serum 25(OH)D concentrations are common across several pediatric disease states, with an overall random-effects standardized mean difference of -1.06 (95% CI -1.67 to -0.45). The direction of effect remained negative in every leave-one-out analysis, suggesting that the association was not driven by a single study. Heterogeneity, however, was very high (I^2=96.3%), so the pooled estimate should be interpreted as a broad pattern rather than a universal effect size.

Clinically, the data indicate that vitamin D insufficiency or deficiency is more frequent among children with selected respiratory, allergic, infectious, and metabolic disorders than among healthy comparators, but the magnitude of difference varies considerably by disease category and study setting.

The clearest quantitative signal in the present synthesis was seen in asthma. All four included asthma studies from Iran, India, Turkey, and India reported lower mean 25(OH)D concentrations in affected children [7-10]. This finding agrees with the earlier meta-analysis by Jat and Khairwa and with the 2024 Arch Public Health review focused on Asia and Africa, both of which concluded that pediatric asthma is frequently accompanied by low vitamin D status [11,12]. A recent Saudi comparative study by Asseri also documented markedly lower vitamin D levels and a substantially higher prevalence of deficiency among asthmatic children than among non-asthmatic controls [25]. Together, these data make low vitamin D one of the most reproducible biochemical associations in childhood asthma research.

Association, however, is not the same as therapeutic efficacy. Observational studies can reflect causal biology, but they can also capture reduced outdoor activity, obesity, corticosteroid exposure, or confounding by lifestyle and socioeconomic variables. Trial evidence remains mixed. The individual participant data meta-analysis by Jolliffe et al. suggested that vitamin D supplementation may reduce severe asthma exacerbations in some populations [23], whereas the 2022 systematic review of randomized trials in childhood asthma concluded that routine supplementation did not clearly prevent exacerbations, emergency visits, or hospitalization [24]. The most plausible interpretation is that benefit, if present, may be restricted to children with baseline deficiency, infection-prone phenotypes, or short-term follow-up windows. Therefore, the present findings support targeted assessment of vitamin D status in high-risk asthmatic children, but they do not justify universal supplementation as disease-specific therapy.

Atopic dermatitis showed a similar, though less precise, pattern. Both the Indian case-control study by Sharma and the Korean pediatric subgroup analyzed by Han reported lower vitamin D levels in affected children, but the pooled confidence interval was wide because of small sample size and marked heterogeneity [13,14]. Even so, the direction of effect is supported by newer evidence. Fu et al. reported lower serum 25(OH)D levels and greater deficiency risk in pediatric atopic dermatitis, while Nielsen et al. showed that supplementation was associated with a modest but significant improvement in disease severity scores [15,16]. These observations are biologically coherent because vitamin D influences keratinocyte differentiation, epidermal barrier integrity, and antimicrobial peptide expression. In practice, this makes vitamin D particularly relevant in children with recurrent, severe, or winter-exacerbated eczema.

Respiratory infection was represented by a large Nigerian case-control study and supported by broader pediatric literature. In our pooled analysis, children with acute respiratory infection had lower vitamin D levels than controls [17]. This direction is consistent with the bronchiolitis study by Golan-Tripto et al. and with the BMJ Open meta-analysis by Cariolou et al., which linked vitamin D deficiency in acutely ill children to worse outcomes [18,19]. The 2024 Endocrine Society guideline similarly acknowledged a possible preventive role for empiric supplementation in reducing respiratory tract infections in children and adolescents [3]. These converging data strengthen the argument that vitamin D has meaningful relevance to pediatric mucosal immunity, even though the optimal treatment threshold is still debated.

The obesity signal in our quantitative synthesis was weaker because the only directly eligible case-control study found little difference between obese and non-obese children [20]. That isolated result should be interpreted cautiously. The broader pediatric evidence, including the 2021 meta-analysis by Fiamenghi and Mello, indicates that obesity is associated with a significantly higher risk of vitamin D deficiency [21]. A likely explanation is that in populations with very high background deficiency, between-group contrasts can narrow even though the overall burden remains substantial. For type 1 diabetes, the evidence is mainly narrative rather than directly poolable, but the 2025 pediatric-focused meta-analysis by Fan et al. found improved 25(OH)D levels and lower insulin requirements after supplementation, while effects on HbA1c remained inconsistent [22]. This again suggests that vitamin D may be clinically relevant without yet qualifying as a stand-alone disease-modifying therapy.

This review has several strengths. It approached vitamin D as a pediatric systems issue rather than a bone-only topic, and it combined narrative synthesis with random-effects pooling, subgroup analysis, leave-one-out sensitivity testing, and Egger regression. The negative pooled effect remained directionally stable throughout these checks. Nonetheless, important limitations remain. The quantitative synthesis was based on observational studies and therefore cannot establish causality. Heterogeneity was extremely high, some subgroups contained only one or two studies, and serum 25(OH)D was generally measured at a single time point. Residual confounding by adiposity, diet, physical activity, sun exposure, season, and medication use is unavoidable. Consequently, the present review should be read as evidence of a consistent association rather than proof that low vitamin D directly causes each pediatric disorder.

The main clinical implication is the need for a risk-stratified rather than indiscriminate approach. Children with recurrent wheezing or asthma, chronic atopic dermatitis, repeated respiratory infections, obesity, limited sun exposure, malabsorption, or autoimmune disease deserve closer attention to vitamin D status within routine pediatric care pathways [2-6].

Prevention remains more evidence-based than late corrective treatment alone: infant supplementation, diet optimization, and safe outdoor activity remain core strategies [2,3,5,6]. Future research should prioritize standardized assays and cut-offs, longitudinal cohorts, and randomized trials restricted to children with verified deficiency. Such work is essential to determine whether vitamin D is primarily a biomarker of pediatric ill health or a modifiable contributor to disease expression and severity.

Conclusion

Current pediatric evidence indicates that lower vitamin D status is consistently associated with several childhood disease states, especially asthma, and supportive evidence also exists for atopic dermatitis, respiratory infection, obesity-related risk, and immune-metabolic dysregulation. The present meta-analysis demonstrates an overall reduction in serum 25(OH)D among diseased children compared with healthy controls, although heterogeneity remains high. These findings support targeted prevention, risk-based screening, and correction of deficiency in vulnerable children, while underscoring the need for better standardized cohort studies and deficiency-targeted randomized trials before broad disease-specific therapeutic claims can be made.

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