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Impact of Vitamin D Levels on Disease Control Across Asthma Subtypes in Mixed-Care Settings

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Abstract

Background: Vitamin D has been implicated in asthma pathophysiology through immunomodulatory and anti-inflammatory mechanisms, although evidence regarding its association with asthma control remains inconsistent. There are no data for the Albanian population that we can use to investigate the level of serum 25(OH)D and its correlation with clinical variables in asthma patients

Methods and Results: This cross-sectional study included 163 adult patients diagnosed with bronchial asthma who were ambulatory, consulted, or hospitalized in the University Hospital Center Tirana, Albania, between November 2024 and October 2025. Asthma control was assessed using the Asthma Control Visual Analogue Scale (AC-VAS; 0-25). Serum 25(OH)D concentration was measured by chemiluminescent immunoassay (CLIA). The primary outcome variable was asthma control measured by the AC-VAS score. Serum 25(OH)D (ng/mL) was analyzed both as a continuous variable and as a categorical variable. Associations were examined using non-parametric tests, linear regression analysis, and moderation analysis (PROCESS macro) to explore potential effect modification by vitamin D status.

Serum 25(OH)D levels were 18.35±8.72 ng/mL, indicating a high prevalence of deficiency. Spearman's rank correlation analysis demonstrated a statistically significant negative correlation between age and serum 25(OH)D levels ($\rho = -0.24$, $P = 0.003$). Serum 25(OH)D levels were positively associated with AC-VAS scores, although this association did not reach statistical significance ($P = 0.097$). Spearman's rank correlation analysis demonstrated a statistically significant negative correlation between AC-VAS scores and age ($\rho = -0.19$, $P = 0.015$), as well as between AC-VAS scores and body mass index (BMI) ($\rho = -0.17$, $P = 0.031$). In the multivariable-adjusted general linear model, age remained independently and negatively associated with AC-VAS scores (B = −0.048, SE = 0.022; $P = 0.030$). Serum 25(OH)D levels (B = 0.039, SE = 0.036; $P = 0.269$) and BMI (B = −0.065, SE = 0.057; $P = 0.253$) were not significantly associated with asthma control after adjustment. Asthma phenotype remained statistically significantly associated with AC-VAS scores in the adjusted model (global test $P = 0.031$). Among the phenotype categories, phenotype 6 was independently associated with higher AC-VAS scores than phenotype 1 (B = 3.612, SE = 1.198; $P = 0.003$). The moderation analysis indicated that the association between BMI and asthma control differed according to 25(OH)D category, with a significant conditional effect observed only at serum 25(OH)D levels ≥ 20 ng/mL.

Conclusion: Vitamin D deficiency was highly prevalent among Albanian adults with asthma but was not independently associated with asthma control. Asthma control was associated with age, body mass index, and asthma phenotype. Vitamin D did not show a direct effect but appeared to modify the association between BMI and asthma control, suggesting a context-dependent role. Further longitudinal and interventional studies are required to confirm these findings and clarify their clinical implications. (**International Journal of Biomedicine. 2026;16(2):201-206.**)

Keywords: vitamin D • asthma control • body mass index • asthma phenotype • moderation analysis

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Introduction

Asthma is a clinically heterogeneous syndrome characterized by chronic airway inflammation and airway hyperresponsiveness, its hallmark features. Distinct aggregations of demographic and clinical attributes have been delineated as "clinical asthma phenotypes," generally showing limited correlation with discrete underlying pathobiological mechanisms or differential therapeutic responsiveness.[1] Vitamin D may influence asthma through effects on lung development, immune regulation, and airway smooth-muscle remodeling. Many studies suggest that lower vitamin D levels are linked to more severe asthma, though results are not always consistent. These potential effects have led to interest in using vitamin D as a preventive or therapeutic option in children with asthma.[2] Recent investigations have identified a correlation between vitamin D deficiency and an elevated risk of both asthma onset and exacerbation.[3,4] Vitamin D deficiency was associated with increased risk of asthma, current wheezing, and reduced lung function in a large study of British adults, suggesting that higher vitamin D levels may reduce asthma risk and improve disease control.[5] A systematic review of randomized controlled trials up to November 2022 examined the effects of vitamin D supplementation in children and adults with asthma. While vitamin D did not reduce type 2 inflammatory markers such as IgE or eosinophils, it was associated with increased serum IL-10, an anti-inflammatory cytokine. These findings suggest that vitamin D may modulate anti-inflammatory pathways rather than directly affecting type 2 inflammation.[6] Biomarkers indicative of type 2-driven airway inflammation have particular clinical utility, especially for phenotyping, stratifying, and managing refractory and severe asthma.

There are no data for the Albanian population that we can use to investigate the level of serum 25(OH)D and its correlation with clinical variables in asthma patients. This study provides evidence for a potential association between serum vitamin D levels and reported asthma control, potentially linked to disease severity, laying the groundwork for future research and informing possible preventive and therapeutic strategies in the Albanian population.

Materials and Methods

This cross-sectional study included 163 adult patients diagnosed with bronchial asthma who were ambulatory, consulted, or hospitalized in the University Hospital Center Tirana, Albania, between November 2024 and October 2025. Demographic and clinical data were collected, including age, gender, body mass index (BMI), smoking status, skin prick test results, comorbidities such as rhinosinusitis, nasal polyposis, and asthma phenotype classification. Asthma control was assessed using the Asthma Control Visual Analogue Scale (AC-VAS; 0-25) (Figure 1).

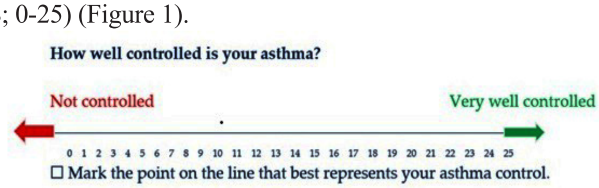

Figure 1. *AC-VAS; 0-25 for asthma control.*

Serum 25(OH)D concentration was measured by chemiluminescent immunoassay (CLIA). Serum 25(OH) D values were available for 155 participants. The primary outcome variable was asthma control measured by the AC-VAS score. Serum 25(OH)D (ng/mL) was analyzed both as a continuous variable and as a categorical variable. For moderation analyses, 25(OH)D status was dichotomized (<20 ng/mL vs ≥20 ng/mL), while for subgroup analyses, 25(OH) D levels were categorized into three groups (<20, 20–30, and >30 ng/mL). Independent variables included age, age at asthma diagnosis, BMI, gender, asthma phenotype, smoking status, rhinosinusitis, nasal polyposis, and skin prick test categories. Asthma clinical phenotypes were classified into six subtypes based on available anamnestic and clinical data (Tables 1 and 2).

Table 1.
Characteristics of classification for probably T2-high phenotypes.

Key Characteristics for likely T2-High phenotypes	
Phenotype 1	Childhood onset; atopy-positive, positive skin allergy tests, ↑ specific IgE
Phenotype 2	Adulthood onset steroid-resistant, blood eosinophilia, sputum eosinophils/neutrophils, CRSwNP
Phenotype 3	Adulthood onset, CRSwNP + NSAID sensitivity

Table 2.
Characteristics of classification for probably T2 -low phenotypes.

Key Characteristics for Likely T2-Low Phenotypes	
Phenotype 4	Middle-aged, non-atopic woman, severe symptoms, perimenopausal
Phenotype 5	Adults, smoking history, steroid resistance, neutrophils in sputum, fixed airflow obstruction
Phenotype 6	Older adults >55 years, non-atopic, severe symptoms, frequent exacerbations, more comorbidities, misdiagnosed as COPD.

Statistical analysis

Continuous variables are reported as mean ± SD and categorical variables as n (%). Due to non-normal distributions, non-parametric tests were used. Spearman's ρ assessed correlations between vitamin D or AC-VAS and continuous variables. The Mann–Whitney U and Kruskal–Wallis tests were used to compare vitamin D and AC-VAS across binary and multi-category clinical variables, respectively. Subgroup analyses by asthma phenotypes examined AC-VAS across 25(OH)D categories (<20, 20–30, >30 ng/mL) using Kruskal–Wallis tests. General linear models were used to assess crude and multivariable-adjusted associations between age, BMI, vitamin D, asthma phenotype, and asthma control (AC-VAS), with Bonferroni correction applied for multiple pairwise comparisons of asthma phenotypes.

Moderation analyses were conducted using PROCESS for SPSS (Model 1) to assess whether 25(OH)D status (<20 ng/mL vs ≥ 20 ng/mL) modified associations between predictors and asthma control (AC-VAS). Age, asthma phenotype, and BMI were tested in separate models. Main effects, predictor × 25(OH)D interactions, and conditional effects were examined.

Results

Among 163 patients, 109(66.9%) were females and 54(33.1%) were males. The mean age was 48.24 ± 16.70 years, while the mean age at asthma diagnosis was 32.88 ± 16.71 years. The mean body mass index (BMI) was 28.26 ± 5.73 kg/m². The mean AC-VAS score was 14.58±3.86. Serum 25(OH)D levels were 18.35 ± 8.72 ng/mL for 155 participants. Regarding asthma phenotype distribution, phenotype 2 was the most prevalent (64/39.3% cases), followed by phenotype 1 (58/35.6% cases). The remaining phenotypes were less frequent. Smokers accounted for 26(16.0%) participants. Rhinosinusitis was present in 74(45.7%) patients, and nasal polyposis in 29(17.8%) patients. Skin prick testing for aeroallergens was negative in 82 (50.3%) patients, while sensitization to only indoor allergens was observed in 32 (19.6%) cases, to only outdoor allergens in 12 (7.4%) cases, and to both indoor and outdoor allergens in 37 (22.7%) cases (Table 3).

Table 3.

Baseline demographic and clinical characteristics of the study population.

Variable	Value
Age (years)	48.24 ± 16.70
Age at diagnosis (years)	32.88 ± 16.71
Body mass index (kg/m²)	28.26 ± 5.73
Serum 25(OH)D (ng/mL)	18.35 ± 8.72 †
Asthma control (AC-VAS, 0–25)	14.58 ± 3.86
Gender	
Female	109 (66.9)
Male	54 (33.1)
Asthma phenotype	
Phenotype 1	58 (35.6)
Phenotype 2	64 (39.3)
Phenotype 3	7 (4.3)
Phenotype 4	9 (5.5)
Phenotype 5	10 (6.1)
Phenotype 6	15 (9.2)
Smoking status	
Yes	26 (16.0)
No	137 (84.0)
Rhinosinusitis	
Yes	74 (45.7)
No	88 (54.3)
Nasal polyposis	
Yes	29 (17.8)
No	134 (82.2)
Skin prick test	
Negative	82 (50.3)
Indoor	32 (19.6)
Outdoor	12 (7.4)
Both	37 (22.7)

Data are presented as mean ± standard deviation or n (%), as appropriate.
† Vitamin D values were available for 155 participants.
AC-VAS: Asthma Control Visual Analogue Scale.

Spearman's rank correlation analysis demonstrated a statistically significant negative correlation between age and serum 25(OH)D levels (ρ = −0.24, *P* = 0.003). No significant correlation was observed between 25(OH)D levels and BMI (ρ = −0.08, *P* = 0.352). Comparisons of gender, asthma phenotype, smoking status, rhinosinusitis, nasal polyposis, and skin prick test categories showed no statistically significant differences in 25(OH)D levels (Table 4).

Table 4.

Associations between serum 25(OH)D levels and demographic and clinical variables.

Variables compared	Statistical test	Test statistic	*P*-value*
Vitamin D and age	Spearman's rank correlation	ρ = −0.24	0.003
Vitamin D and BMI	Spearman's rank correlation	ρ = −0.08	0.352
Vitamin D across sex (male vs female)	Mann–Whitney U test	Z = 0.63	0.532
Vitamin D across asthma phenotype	Kruskal–Wallis test	H = 3.07	0.690
Vitamin D across smoking status	Mann–Whitney U test	Z = 0.25	0.805
Vitamin D across rhinosinusitis	Mann–Whitney U test	Z = −0.50	0.619
Vitamin D across nasal polyposis	Mann–Whitney U test	Z = −0.70	0.483
Vitamin D across prick test categories	Kruskal–Wallis test	H = 0.21	0.976

** P-values derived from Spearman's rank correlation, Mann–Whitney U test, or Kruskal–Wallis test. A P-value of < 0.05 was considered statistically significant.*

Spearman's rank correlation analysis demonstrated a statistically significant negative correlation between AC-VAS scores and age (ρ = −0.19, *P* = 0.015), as well as between AC-VAS scores and BMI (ρ = −0.17, *P* = 0.031). No significant correlation was observed between AC-VAS scores and age at diagnosis (ρ = −0.04, *P* = 0.641) or serum 25(OH)D levels (ρ = 0.14, *P* = 0.095).

Comparisons across gender, smoking status, rhinosinusitis, nasal polyposis, and skin prick test categories showed no statistically significant differences in AC-VAS scores (all *P* > 0.05). In contrast, a statistically significant difference in AC-VAS scores was observed across asthma phenotypes (H = 13.11, *P* = 0.022). In subgroup analyses stratified by asthma phenotypes, no statistically significant differences in AC-VAS scores were observed across 25(OH)D categories in either likely T2-high or likely T2-low phenotypes (Table 5).

In crude (unadjusted) analyses, increasing age was significantly associated with lower AC-VAS scores (B = −0.042, SE = 0.018; *P* = 0.021). BMI also showed a significant negative association with asthma control (B = −0.110, SE = 0.052; *P*= 0.037). Serum 25(OH)D levels were positively associated with AC-VAS scores, although this association did not reach statistical significance (B = 0.060, SE = 0.036; *P* = 0.097). In the unadjusted model, asthma phenotypes showed a significant overall association with AC-VAS scores, as indicated by the global test (*P* = 0.026), with phenotype 6 demonstrating higher AC-VAS scores than phenotype 1.

Table 5.

Associations between asthma control (AC-VAS) and demographic and clinical variables.

Variables compared	Statistical test	Test statistic	*P*-value*
AC-VAS and age	Spearman's rank correlation	ρ = −0.19	0.015
AC-VAS and age at diagnosis	Spearman's rank correlation	ρ = −0.04	0.641
AC-VAS and BMI	Spearman's rank correlation	ρ = −0.17	0.031
AC-VAS and Vit D	Spearman's rank correlation	ρ = 0.14	0.095
AC-VAS across sex (male vs female)	Mann–Whitney U test	Z = 0.86	0.388
AC-VAS across asthma phenotype	Kruskal–Wallis test	H = 13.11	0.022
AC-VAS across smoking status	Mann–Whitney U test	Z = −0.33	0.742
AC-VAS across rhinosinusitis	Mann–Whitney U test	Z = −1.15	0.252
AC-VAS across prick test categories	Kruskal–Wallis test	H = 7.06	0.070
AC-VAS across nasal polyposis	Mann–Whitney U test	Z = −0.27	0.787
AC-VAS across Vit D categories in T_2- high asthma phenotypes	Kruskal–Wallis test	H = 1.98	0.372
AC-VAS across Vit D categories in T_2-low asthma phenotypes	Kruskal–Wallis test	H = 1.75	0.418

** P-values derived from Spearman's rank correlation, Mann–Whitney U test, or Kruskal–Wallis test. A P-value of < 0.05 was considered statistically significant.*

In the multivariable-adjusted general linear model, age remained independently and negatively associated with AC-VAS scores (B = −0.048, SE = 0.022; *P* = 0.030). Serum 25(OH)D levels (B = 0.039, SE = 0.036; *P* = 0.269) and BMI (B = −0.065, SE = 0.057; *P* = 0.253) were not significantly associated with asthma control after adjustment. Asthma phenotype remained statistically significantly associated with AC-VAS scores in the adjusted model (global test *P* = 0.031). Among the phenotype categories, phenotype 6 was independently associated with higher AC-VAS scores than phenotype 1 (B = 3.612, SE = 1.198; *P* = 0.003) (Table 6).

No statistically significant interaction effects were observed for age or asthma phenotype. A statistically significant conditional effect of BMI on AC-VAS scores was observed in participants with serum 25(OH)D levels ≥ 20 ng/mL (b = −0.22, *P* = 0.010), whereas no significant conditional effects of BMI were found in participants with 25(OH)D levels < 20 ng/mL (all *P* > 0.05). (Table 7)

Serum 25(OH)D did not show an independent association with asthma control. However, the moderation analysis indicated that the association between BMI and asthma control differed according to 25(OH)D category, with a significant conditional effect observed only at serum 25(OH) D levels ≥ 20 ng/mL (Figure 2).

Table 6.

Crude and multivariable-adjusted generalized linear model (GLM) analyses of factors associated with asthma control (AC-VAS).

Predictor	Crude B (SE)	*p*-value	Adjusted B (SE)	*P*-value
Age	-0.042 (0.018)	0.021	-0.048 (0.022)	0.030
Serum 25(OH)D	0.060 (0.036)	0.097	0.039 (0.036)	0.269
BMI	-0.110 (0.052)	0.037	-0.065 (0.057)	0.253
Asthma phenotype (global test)	—	0.026	—	0.031
Phenotype 2	0.474 (0.682)	0.488	1.097 (0.733)	0.137
Phenotype 3	-2.591 (1.505)	0.087	-0.757 (1.670)	0.651
Phenotype 4	-2.448 (1.348)	0.071	-0.766 (1.453)	0.599
Phenotype 5	-0.148 (1.288)	0.908	1.093 (1.399)	0.436
Phenotype 6	2.218 (1.090)	0.043	3.612 (1.198)	0.003
Phenotype 1	Reference	—	Reference	—

Two-tailed P-values are reported. A P-value of < 0.05 was considered statistically significant.

Table 7.

Moderation analysis of the association between selected predictors and asthma control (AC-VAS) by vitamin D category (PROCESS Model 1).

Focal predictor (X)	n	Main effect on AC-VAS	Interaction X × Vit D status	Conditional effects of X on AC-VAS
Age	155	b = −0.04 *P* = 0.030	b = 0.04 *P* = 0.266	Conditional effects not reported; no evidence of differential effects by vitamin D category
Asthma phenotype	155	b = −0.24 *P* = 0.762	b = 0.26 *P* = 0.876	Conditional effects not reported; no evidence of conditional effects
BMI	154	b = −0.10 *P* = 0.055	b = −0.19 *P* = 0.082	Vitamin D <20 ng/mL: b = −0.03, *P* = 0.673 Vitamin D ≥20ng/mL: b = −0.22, *P*= 0.010

A P-value of < 0.05 was considered statistically significant.

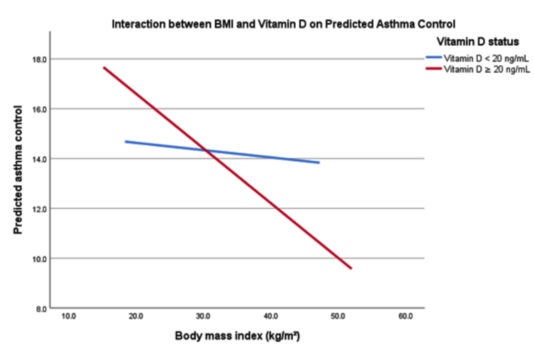

Figure 2. *Predicted AC-VAS scores across BMI levels stratified by vitamin D category.*

Discussion

In this cross-sectional study of Albanian adults with asthma, serum 25(OH)D levels were generally low, with mean concentrations in the deficient range, confirming a high prevalence of suboptimal serum 25(OH)D status in this population. This finding is consistent with reports from other European cohorts and may reflect limited sun exposure, dietary habits, or lifestyle factors.[7,8] Serum 25(OH)D levels were inversely associated with age, consistent with previous evidence that advancing age is associated with reduced vitamin D synthesis and availability.[9]

Despite widespread deficiency, serum 25(OH)D was not independently associated with reported asthma control, as assessed by the AC-VAS. The lack of a direct association persisted across correlation analyses, subgroup comparisons, and multivariable-adjusted regression models. These findings align with several randomized trials and observational studies suggesting that vitamin D may not exert a strong direct effect on symptom control, particularly when assessed cross-sectionally.[10]

In contrast, age and BMI emerged as more consistent determinants of asthma control, with older age and higher BMI associated with poorer control. These findings are clinically relevant, with BMI modifiable or addressable through targeted interventions. In adjusted models, asthma phenotype was associated with asthma control; notably, phenotype 6 was independently associated with higher AC-VAS scores than phenotype 1. Serum 25(OH)D levels did not differ significantly across phenotypes, suggesting comparable serum 25(OH)D status across clinical asthma subtypes.

An important and novel observation of this study was the moderating role of vitamin D on the relationship between BMI and asthma control. The negative association between BMI and asthma control was evident only among participants with serum 25(OH)D levels $\geq$ 20 ng/mL, whereas no significant association was observed in those with vitamin D deficiency. This finding suggests that serum 25(OH) D status may modify the impact of adiposity on asthma-related symptoms, potentially through immunomodulatory or metabolic pathways. Although the interaction term reached only borderline statistical significance, the conditional effects analysis supports a biologically plausible modifying role of vitamin D rather than a direct effect on disease control.

Contrary to some prior studies, serum 25(OH)D was not associated with asthma subtype, smoking status, rhinosinusitis, nasal polyposis, or allergic sensitization patterns. This may reflect population-specific factors, limited statistical power for subgroup analyses, or the multifactorial nature of asthma control, in which vitamin D plays a secondary or context-dependent role.

Overall, these findings suggest that serum 25(OH)D deficiency is highly prevalent in Albanian asthma patients but does not independently determine asthma control. Instead, vitamin D may act as a contextual modifier, influencing the relationship between metabolic factors, such as BMI, and asthma outcomes. Longitudinal studies and randomized controlled trials are needed to determine whether vitamin D supplementation improves asthma-related outcomes in selected subgroups, particularly overweight or obese patients.

Limitations

It should be noted that cross-sectional design limits causal interpretation, and vitamin D was assessed at a single time point. Residual confounding and limited power in subgroup analyses cannot be excluded.

Conclusion

Vitamin D deficiency was highly prevalent among Albanian adults with asthma but was not independently associated with asthma control. Asthma control was associated with age, body mass index, and asthma phenotype. Vitamin D did not show a direct effect but appeared to modify the association between BMI and asthma control, suggesting a context-dependent role. Further longitudinal and interventional studies are required to confirm these findings and clarify their clinical implications.

Ethical Statement

This study was approved by the Ethical Committee at the University of Medicine (Tirana) with decision no. 29, dated 29.08.2024, and informed consent was obtained from each participant.

Author Contributions

Mehmet Hoxha: Conceptualization, Methodology, Formal analysis, Investigation, Writing – original draft, Supervision.
Eralda Lekli: Methodology, Investigation, Data curation, Writing – review and editing.
Dorian Shkembi: Investigation, Data curation.
Ester Ndreu: Investigation, Data Curation.
Xhein Hajrulla: Investigation, Data curation.
Etleva Qirko: Supervision, Writing – review and editing.
Mehmet Hoxha is the primary author of this work.
All authors reviewed and approved the final manuscript.

Conflict of Interest

The authors have declared no conflict of interest.

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Data Availability Statement

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

References

1. Status Asthmaticus Medication: Anticholinergics, Respiratory, Beta2 Agonists, Corticosteroids, Electrolytes, Monoclonal Antibodies, Anti-asthmatics, Xanthine Derivatives [Internet]. [cited 2026 Jan 23]. Available from: https://emedicine.medscape.com/article/2129484-medication?form=fpf

2. Salmanpour F, Kian N, Samieefar N, Khazeei Tabari MA, Rezaei N. Asthma and Vitamin D Deficiency: Occurrence, Immune Mechanisms, and New Perspectives. J Immunol Res. 2022 Jul 15;2022:6735900. doi: 10.1155/2022/6735900. PMID: 35874901; PMCID: PMC9307373.

3. Ogeyingbo OD, Ahmed R, Gyawali M, Venkatesan N, Bhandari R, Botleroo RA, Kareem R, Elshaikh AO. The Relationship Between Vitamin D and Asthma Exacerbation. Cureus. 2021 Aug 18;13(8):e17279. doi: 10.7759/cureus.17279. PMID: 34462708; PMCID: PMC8389855.

4. Wang Q, Ying Q, Zhu W, Chen J. Vitamin D and asthma occurrence in children: A systematic review and meta-analysis. J Pediatr Nurs. 2022 Jan-Feb;62:e60-e68. doi: 10.1016/j.pedn.2021.07.005. Epub 2021 Aug 6. PMID: 34366195.

5. Zhu Y, Jing D, Liang H, Li D, Chang Q, Shen M, Pan P, Liu H, Zhang Y. Vitamin D status and asthma, lung function, and hospitalization among British adults. Front Nutr. 2022 Aug 10;9:954768. doi: 10.3389/fnut.2022.954768. PMID: 36034921; PMCID: PMC9399919.

6. El Abd A, Dasari H, Dodin P, Trottier H, Ducharme FM. The effects of vitamin D supplementation on inflammatory biomarkers in patients with asthma: a systematic review and meta-analysis of randomized controlled trials. Front Immunol. 2024 Mar 13;15:1335968. doi: 10.3389/fimmu.2024.1335968. PMID: 38545098; PMCID: PMC10965564.

7. Cashman KD, Dowling KG, Škrabáková Z, Gonzalez-Gross M, Valtueña J, De Henauw S, et al. Vitamin D deficiency in Europe: pandemic? Am J Clin Nutr. 2016 Apr;103(4):1033-44. doi: 10.3945/ajcn.115.120873. Epub 2016 Feb 10. PMID: 26864360; PMCID: PMC5527850.

8. Spiro A, Buttriss JL. Vitamin D: An overview of vitamin D status and intake in Europe. Nutr Bull. 2014 Dec;39(4):322-350. doi: 10.1111/nbu.12108. PMID: 25635171; PMCID: PMC4288313.

9. Giustina A, Bouillon R, Dawson-Hughes B, Ebeling PR, Lazaretti-Castro M, Lips P, Marcocci C, Bilezikian JP. Vitamin D in the older population: a consensus statement. Endocrine. 2023 Jan;79(1):31-44. doi: 10.1007/s12020-022-03208-3. Epub 2022 Oct 26. PMID: 36287374; PMCID: PMC9607753.

10. Williamson A, Martineau AR, Sheikh A, Jolliffe D, Griffiths CJ. Vitamin D for the management of asthma. Cochrane Database Syst Rev. 2023 Feb 6;2(2):CD011511. doi: 10.1002/14651858.CD011511.pub3. PMID: 36744416; PMCID: PMC9899558.

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