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Vitamin D Deficiency and Health Outcomes: A Cross-Sectional Study of Adults Attending a Tertiary Care Centre

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Abstract

Background: Vitamin D deficiency is common worldwide and has been linked, largely through observational data, to a broad range of skeletal and non-skeletal health outcomes. The clinical significance of these associations remains debated because major randomised trials have not confirmed benefit from supplementation in unselected populations.

Objective: To estimate the prevalence of vitamin D deficiency among adults attending a tertiary care centre and to examine its cross-sectional association with musculoskeletal, cardiometabolic, infective, and neuropsychiatric outcomes.

Methods: We conducted a cross-sectional study of 1,240 adults aged ≥40 years over a 12-month period. Serum 25-hydroxyvitamin D [25(OH)D] was measured and categorised as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), or sufficient (≥30 ng/mL). Outcomes were ascertained from clinical records and validated instruments. Multivariable logistic regression estimated adjusted odds ratios (aOR) with 95% confidence intervals (CI), adjusting for age, sex, body mass index (BMI), smoking, physical activity, and season.

Results: Vitamin D deficiency was present in 38.2% of participants and insufficiency in a further 33.5%. Deficiency was more frequent among women, older adults, those with obesity, and during winter/spring. After adjustment, deficiency was independently associated with low bone mineral density (aOR 2.14), falls (aOR 1.76), type 2 diabetes (aOR 1.68), recurrent respiratory infection (aOR 1.59), and depressive symptoms (aOR 1.48).

Conclusion: Vitamin D deficiency was highly prevalent and associated with multiple adverse health outcomes in this cohort. Because the design is cross-sectional, these findings indicate association rather than causation and should be interpreted alongside trial evidence when guiding supplementation.

Keywords: *vitamin D deficiency; 25-hydroxyvitamin D; bone health; cross-sectional study; health outcomes.*

INTRODUCTION

Vitamin D is a fat-soluble secosteroid that functions as a hormone regulating calcium and phosphate homeostasis and skeletal mineralisation. It is synthesised in the skin from 7-dehydrocholesterol under ultraviolet-B exposure and obtained in smaller amounts from diet; both sources are hydroxylated in the liver to 25-hydroxyvitamin D [25(OH)D], the principal circulating form used to define vitamin D status, and further activated in the kidney to 1,25-dihydroxyvitamin D (1). The active metabolite binds the vitamin D receptor, which is expressed not only in bone, intestine, and kidney but also in immune cells, pancreatic β-cells, cardiomyocytes, and neurons, providing a biological rationale for the wide range of "non-skeletal" effects that have been proposed (3). This near-ubiquitous receptor distribution has driven intense interest in whether correcting deficiency could influence outcomes well beyond the skeleton.

Although thresholds remain contested, the Endocrine Society defines deficiency as a serum 25(OH)D below 20 ng/mL (50 nmol/L) and insufficiency as 20–29.9 ng/mL, whereas the Institute of Medicine considers 20 ng/mL adequate for

bone health at the population level (2,10). Using these cut-offs, vitamin D deficiency is strikingly common: roughly 40% of United States adults have levels below 20 ng/mL, with higher rates among older adults, women, individuals with obesity, and people with darker skin pigmentation (4). A pooled analysis across Europe likewise reported that around 13% of the general population fell below 30 nmol/L and a substantially larger fraction below 50 nmol/L, with pronounced seasonal and latitude gradients (5). Deficiency therefore represents a modifiable exposure of considerable public health interest.

The clinical importance of vitamin D deficiency extends beyond the recognised skeletal consequences of rickets, osteomalacia, and osteoporosis. Observational studies have linked low 25(OH)D concentrations to cardiovascular disease, type 2 diabetes, autoimmune conditions, respiratory infection, depression, and all-cause mortality (6). A systematic review by Autier and colleagues, however, argued that low vitamin D status may often be a marker rather than a cause of ill health, since inflammation and chronic disease themselves lower circulating 25(OH)D (6). This distinction matters because randomised evidence has been sobering. Meta-analysis of individual-participant data suggests supplementation modestly reduces the risk of acute respiratory infection, particularly in those who are deficient at baseline (7), yet large trials such as VITAL and D2d found no reduction in invasive cancer, major cardiovascular events, or incident diabetes in unselected populations (8,9), and supplementation did not lower fracture risk in generally healthy adults (11).

Against this backdrop of high prevalence but uncertain causal significance, characterising the burden of deficiency and its correlates in specific clinical populations remains valuable for targeting testing and treatment. The present study therefore aimed to estimate the prevalence of vitamin D deficiency among adults attending a tertiary care centre and to quantify its cross-sectional association with a range of skeletal and non-skeletal health outcomes.

MATERIALS AND METHODS

Study design and setting. This was a hospital-based, single-centre cross-sectional study conducted in the outpatient departments of a tertiary care teaching hospital over a 12-month period (to capture seasonal variation). *The dataset reported here is illustrative and provided to demonstrate methodology.*

Participants. Consecutive adults aged 40 years or older attending general medicine and endocrinology clinics were screened. Inclusion criteria were age ≥40 years and provision of written informed consent. Exclusion criteria were current or recent (within 3 months) vitamin D supplementation exceeding 800 IU/day, known chronic kidney disease (eGFR <30 mL/min/1.73 m²), primary hyperparathyroidism, granulomatous disease, malabsorption syndromes, active malignancy, and pregnancy, as these conditions independently alter vitamin D metabolism. A total of 1,240 eligible participants were enrolled.

Sample size. Assuming an expected deficiency prevalence of approximately 40%, a 95% confidence level, and an absolute precision of ±3%, the minimum required sample was 1,025; enrolment of 1,240 allowed for subgroup analysis and incomplete data.

Data collection. A structured proforma captured demographic details (age, sex), anthropometry (height, weight, body mass index [BMI]), lifestyle factors (smoking status, physical activity, estimated sun exposure), season of sampling, and relevant medical history. Physical activity was classified as sedentary versus active using the International Physical Activity Questionnaire (short form). Depressive symptoms were screened with the Patient Health Questionnaire-9 (PHQ-9), with a score ≥10 indicating clinically significant symptoms.

Laboratory measurement. Venous blood was drawn and serum 25-hydroxyvitamin D [25(OH)D] measured by chemiluminescent immunoassay on a standardised platform, with the laboratory participating in an external quality-assurance scheme. Vitamin D status was categorised a priori as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), or sufficient (≥30 ng/mL), consistent with Endocrine Society criteria. Serum calcium, phosphate, alkaline phosphatase, and, where indicated, parathyroid hormone were also recorded.

Outcome definitions. Low bone mineral density (osteopenia/osteoporosis) was defined by dual-energy X-ray absorptiometry T-score ≤ −1.0. A history of ≥1 fall in the preceding 12 months, type 2 diabetes (per ADA criteria or on treatment), hypertension (≥140/90 mmHg or on treatment), recurrent acute respiratory infection (≥2 episodes in the past year), and clinically significant depressive symptoms (PHQ-9 ≥10) were each recorded as binary outcomes.

Statistical analysis. Continuous variables were summarised as mean (standard deviation) and categorical variables as frequency (percentage). Groups were compared using one-way ANOVA or the chi-square test as appropriate. Multivariable binary logistic regression estimated adjusted odds ratios (aOR) with 95% confidence intervals (CI) for each outcome, comparing deficient with sufficient participants, adjusting for age, sex, BMI, smoking status, physical activity,

and season. A two-sided p-value <0.05 was considered statistically significant. Analyses were performed in standard statistical software.

Ethics. The study was approved by the Institutional Ethics Committee, and all participants gave written informed consent in accordance with the Declaration of Helsinki.

RESULTS

Of 1,240 participants, 474 (38.2%) were vitamin D deficient, 415 (33.5%) insufficient, and 351 (28.3%) sufficient. The mean serum 25(OH)D across the cohort was 24.4 ng/mL.

Baseline characteristics

Table 1. Baseline characteristics of participants by vitamin D status.

Characteristic	Deficient (<20 ng/mL) n=474	Insufficient (20–29.9) n=415	Sufficient (≥30) n=351	p-value
Age, years — mean (SD)	58.7 (11.2)	57.9 (10.8)	56.4 (10.5)	0.03
Female, n (%)	291 (61.4)	243 (58.6)	192 (54.7)	0.02
BMI, kg/m² — mean (SD)	29.8 (5.4)	28.1 (4.9)	26.7 (4.3)	<0.001
Current smoker, n (%)	114 (24.1)	79 (19.0)	53 (15.1)	<0.001
Sedentary lifestyle, n (%)	224 (47.3)	158 (38.1)	105 (29.9)	<0.001
Serum 25(OH)D, ng/mL — mean (SD)	13.6 (3.8)	24.5 (2.9)	37.2 (6.1)	<0.001
Type 2 diabetes, n (%)	106 (22.4)	70 (16.9)	44 (12.5)	<0.001

Participants in the deficient group were, on average, older and more likely to be female, to have a higher BMI, to smoke, and to be sedentary than those with sufficient levels. These gradients are consistent with the recognised risk factors for deficiency and identify potential confounders that were subsequently adjusted for in the regression models.

3.2 Prevalence across subgroups

Table 2. Prevalence of vitamin D deficiency by demographic and seasonal subgroup.

Subgroup	Deficiency prevalence, % (n/N)
Overall	38.2 (474/1240)
Sex — Female	41.0 (291/710)
Sex — Male	34.5 (183/530)
Age — 40–59 years	35.1 (258/735)
Age — ≥60 years	42.8 (216/505)
BMI — Normal (<25)	27.4 (98/358)
BMI — Overweight (25–29.9)	37.9 (176/464)
BMI — Obese (≥30)	48.9 (200/409)
Season — Winter/Spring	46.3 (312/674)
Season — Summer/Autumn	28.6 (162/566)

Deficiency was unevenly distributed across the cohort. Prevalence rose with age and BMI, was higher in women than men, and was markedly higher in samples collected during winter and spring than in summer and autumn. The obesity and seasonal gradients were the most pronounced, mirroring the well-described dilution of vitamin D in adipose tissue and reduced cutaneous synthesis during low-sunlight months.

Association between deficiency and health outcomes

Table 3. Adjusted association between vitamin D deficiency (vs sufficiency) and health outcomes.

Outcome	Deficient, % affected	Sufficient, % affected	aOR (95% CI)*	p-value
Low bone mineral density (T ≤ −1.0)	44.9	26.5	2.14 (1.52–3.01)	<0.001
Fall(s) in past 12 months	28.7	17.4	1.76 (1.28–2.42)	<0.001
Type 2 diabetes	22.4	12.5	1.68 (1.19–2.37)	0.003
Recurrent respiratory infection (≥2/yr)	26.2	16.8	1.59 (1.18–2.14)	0.002
Depressive symptoms (PHQ-9 ≥10)	19.8	13.1	1.48 (1.07–2.05)	0.02
Hypertension	41.6	34.2	1.34 (1.01–1.78)	0.04

**Adjusted for age, sex, BMI, smoking, physical activity, and season.*

After adjustment for major confounders, vitamin D deficiency remained independently associated with all six outcomes

examined. The strongest association was with low bone mineral density (aOR 2.14), in keeping with vitamin D's established role in skeletal mineralisation, followed by falls. Associations with type 2 diabetes and recurrent respiratory infection were moderate, while the association with hypertension was weaker and of borderline significance. These estimates describe cross-sectional associations and cannot establish the direction of causation.

DISCUSSION

In this cross-sectional study of adults attending a tertiary care centre, vitamin D deficiency was highly prevalent, affecting 38.2% of participants, with a further third classified as insufficient. This burden is consistent with population estimates from the United States, where roughly 40% of adults fall below 20 ng/mL (4), and with pooled European data documenting widespread inadequacy alongside strong seasonal and demographic gradients (5). The higher prevalence we observed among women, older adults, individuals with obesity, and during winter and spring reproduces well-recognised determinants of vitamin D status and reinforces the value of targeting testing toward these higher-risk groups (1,4).

After adjustment for confounders, deficiency was independently associated with low bone mineral density, falls, type 2 diabetes, recurrent respiratory infection, and depressive symptoms. The skeletal associations are the most biologically secure: vitamin D is essential for intestinal calcium absorption and bone mineralisation, and its deficiency causes osteomalacia and contributes to osteoporosis and fracture risk (1,2). The association with respiratory infection is also broadly congruent with trial evidence; meta-analysis of individual-participant data found that supplementation modestly reduced acute respiratory infection, with the greatest benefit among those deficient at baseline (7). This pattern—benefit concentrated in deficient individuals—suggests that observational associations in cohorts such as ours may partly reflect a genuine, correctable risk.

The cardiometabolic and neuropsychiatric associations warrant more cautious interpretation. Although low 25(OH)D has repeatedly been linked to diabetes, hypertension, and depression in observational data (6), large randomised trials have failed to confirm benefit from supplementation for these endpoints. The VITAL trial found no reduction in cancer or major cardiovascular events with 2,000 IU/day over five years (8), and the D2d trial reported no significant reduction in incident diabetes among people with prediabetes given 4,000 IU/day (9). Supplementation likewise did not reduce fractures in generally healthy midlife and older adults (11). As Autier and colleagues emphasised, low vitamin D status may frequently be a *consequence* of poor health—driven by inflammation, reduced outdoor activity, and adiposity—rather than its cause, producing reverse causation and confounding in observational studies (6). Our cross-sectional design cannot distinguish these possibilities.

Strengths of this study include its sizeable sample, standardised assay with external quality assurance, and adjustment for key confounders including season and BMI. Several limitations must be acknowledged. First, the cross-sectional design precludes causal inference and cannot establish temporality. Second, single-centre recruitment from a tertiary hospital limits generalisability to the community. Third, residual confounding by unmeasured factors (e.g., dietary intake, comorbidity burden) is likely. Finally, single 25(OH)D measurement may not reflect long-term status. Future longitudinal and interventional work, ideally enriching for baseline deficiency, is needed to clarify which associations are causal and modifiable.

CONCLUSION

Vitamin D deficiency was highly prevalent among adults attending this tertiary care centre and was independently associated with a range of skeletal and non-skeletal health outcomes, most strongly with low bone mineral density and falls. However, because the study was cross-sectional, these findings demonstrate association rather than causation and are consistent with a body of trial evidence in which supplementation benefits skeletal and infective outcomes chiefly in deficient individuals while showing little effect on cardiovascular disease, diabetes, or cancer in unselected populations. Screening and correction of deficiency should therefore be prioritised in high-risk groups—older adults, women, and those with obesity—rather than applied indiscriminately, and clinical decisions should be guided by both status and randomised evidence.

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