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Effects of Vitamin D Supplementation on Obesity and Its Metabolic Complications: A Narrative Review

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Abstract

Background.

Obesity and metabolic syndrome are major contributors to global cardiometabolic morbidity and mortality. Vitamin D deficiency is highly prevalent in individuals with obesity and has been implicated in adverse metabolic regulation. However, evidence regarding the efficacy of vitamin D supplementation in improving obesity-related outcomes remains inconsistent.

Aim.

The aim of this review was to assess current evidence regarding the effects of vitamin D supplementation on obesity and its metabolic complications.

Review methods

A narrative literature review was conducted using electronic searches of the PubMed database, supplemented by reports from the World Health Organization and other peer-reviewed

sources. Preference was given to systematic reviews, meta-analyses, randomized controlled trials, and high-quality observational studies published in English between 2010 and 2024.

Results.

Evidence from randomized controlled trials and meta-analyses consistently demonstrates that vitamin D supplementation increases serum 25-hydroxyvitamin D concentrations in individuals with overweight and obesity. However, its effects on obesity-related metabolic outcomes remain limited and inconsistent. Most studies report no significant improvements in insulin sensitivity, glycemic control, blood pressure, or overall body weight. Some trials have observed modest reductions in body fat mass, body mass index, or waist circumference. Small improvements in selected lipid parameters and inflammatory biomarkers have also been reported, although these findings are not consistently replicated. Potential metabolic benefits appear more pronounced in individuals with baseline vitamin D deficiency.

Conclusions.

Current evidence does not support vitamin D supplementation as an effective standalone therapy for obesity or its major metabolic complications. Its clinical role appears primarily adjunctive, particularly in individuals with confirmed vitamin D deficiency.

Key words: “vitamin D,” “vitamin D supplementation,” “cholecalciferol,” “ergocalciferol,” “obesity,” “overweight,” “metabolic syndrome,” “insulin resistance,” “body composition,” “adiposity,” “fat mass,” “waist circumference,” “vitamin D receptor,” “inflammation,” “energy metabolism,” “randomized controlled trial,” “meta-analysis,” “weight management”

Introduction

Obesity is a chronic multifactorial disease characterized by abnormal or excessive accumulation of body fat. In adults, obesity is most commonly classified using body mass index (BMI), calculated as weight in kilograms divided by height in meters squared (kg/m²), with a BMI of ≥30 kg/m² considered obese. According to the WHO European Regional Obesity Report (“New WHO Report: Europe Can Reverse Its Obesity ‘epidemic’” 2022), in 2022 approximately 59% of adults in the WHO European Region were overweight or living with obesity, indicating epidemic proportions. Obesity correlates with increased risk of metabolic, cardiovascular, musculoskeletal, and certain oncological diseases, and its development results from complex interactions

among genetic, behavioral, environmental, and metabolic factors ("Obesity and Overweight" 202).

Category	**BMI (kg/m²)**
Underweight	< 18.5
Normal weight	18.5–24.9
Overweight (pre-obesity)	25.0-29.0
Obesity class I	30.0–34.9
Obesity class II	35.0-39.9
Obesity class III	≥40.0

Table 1. Classification of Obesity in Adults Based on BMI (World Health Organization. Obesity and overweight. Geneva: WHO; 2024.) https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

Obesity is increasingly recognized as a state of chronic low-grade systemic inflammation. This is reflected by elevated circulating inflammatory biomarkers. Expansion of adipose tissue, particularly within the visceral compartment, is accompanied by infiltration of macrophages and other immune cells, resulting in enhanced secretion of pro-inflammatory cytokines and adipokines. Among the most consistently elevated markers in individuals with obesity are C-reactive protein (CRP), serum amyloid A (SAA), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6). In parallel, dysregulated adipokine profiles—characterized by increased leptin and reduced adiponectin concentrations—further exacerbate metabolic disturbances. Collectively, these inflammatory mediators contribute to the pathogenesis of insulin resistance, endothelial dysfunction, and heightened cardiometabolic risk. Accordingly, circulating inflammatory markers are widely employed as indicators of obesity-related

metabolic complications and as surrogate endpoints in interventional studies (Ellulu et al. 2017).

Obesity is associated with a wide spectrum of adverse health consequences that increase morbidity and mortality worldwide. Excess adipose tissue promotes chronic low-grade inflammation, insulin resistance, dyslipidemia, and hormonal dysregulation. Individuals with obesity are at markedly higher risk of type 2 diabetes, hypertension, coronary artery disease, stroke, and nonalcoholic fatty liver disease. In addition, obesity increases the likelihood of certain cancers, including colorectal, breast (postmenopausal), and endometrial cancer. Beyond metabolic and cardiovascular complications, obesity is linked to musculoskeletal disorders such as osteoarthritis, impaired respiratory function including Obstructive Sleep Apnea, reduced quality of life, and increased all-cause mortality. Given its multifactorial etiology and systemic consequences, obesity represents a major public health challenge requiring comprehensive prevention and management strategies ("Obesity and Overweight" 2024).

Vitamin D is a secosteroid hormone that plays a key role in maintaining calcium and phosphate homeostasis and ensuring proper bone mineralization. Beyond its classical skeletal functions, vitamin D exerts pleiotropic effects in numerous extra-skeletal tissues through activation of the vitamin D receptor (VDR), which is expressed in adipose tissue, pancreatic β-cells, skeletal muscle, and immune cells. Through these pathways, vitamin D participates in the regulation of insulin secretion and sensitivity, modulation of inflammatory responses, and control of cellular proliferation and differentiation. Experimental evidence also suggests that vitamin D may influence adipogenesis and energy metabolism, providing a potential mechanistic link between vitamin D status and obesity-related metabolic disturbances ("National Institutes of Health, Office of Dietary Supplements, Vitamin D," n.d.)

Vitamin D deficiency constitutes a significant global public health concern affecting populations across all age groups and geographic regions. It is most commonly defined as a serum 25-hydroxyvitamin D [25(OH)D] concentration below 20 ng/mL (50 nmol/L), although threshold values differ slightly among professional guidelines. Across Europe, hypovitaminosis D remains highly prevalent, largely attributable to limited ultraviolet B exposure, pronounced seasonal variation, predominantly indoor lifestyles, and insufficient dietary intake. Notably, individuals with obesity exhibit an increased risk of vitamin D deficiency, a phenomenon partly explained by volumetric dilution and sequestration of this

fat-soluble vitamin within adipose tissue, which reduces its circulating bioavailability. Suboptimal vitamin D status has been implicated in disturbances of glucose homeostasis, insulin resistance, low-grade systemic inflammation, and elevated cardiometabolic risk. Collectively, these observations have intensified interest in vitamin D supplementation as a potential adjunctive strategy in the management of obesity and related metabolic disorders (Vranić et al. 2019).

Vitamin D dosing depends on baseline status, body composition, and clinical context. In adults, maintenance supplementation typically ranges from 800 to 2,000 IU/day to achieve adequate serum 25-hydroxyvitamin D [25(OH)D] levels. Individuals with obesity often require higher doses due to volumetric dilution and sequestration of this fat-soluble vitamin in adipose tissue, with some guidelines recommending two- to three-fold greater intake. In cases of deficiency, loading regimens (e.g., 50,000 IU weekly for 6–8 weeks) may be used, followed by maintenance therapy. Supplementation should be individualized and monitored to avoid toxicity (Płudowski et al. 2023).

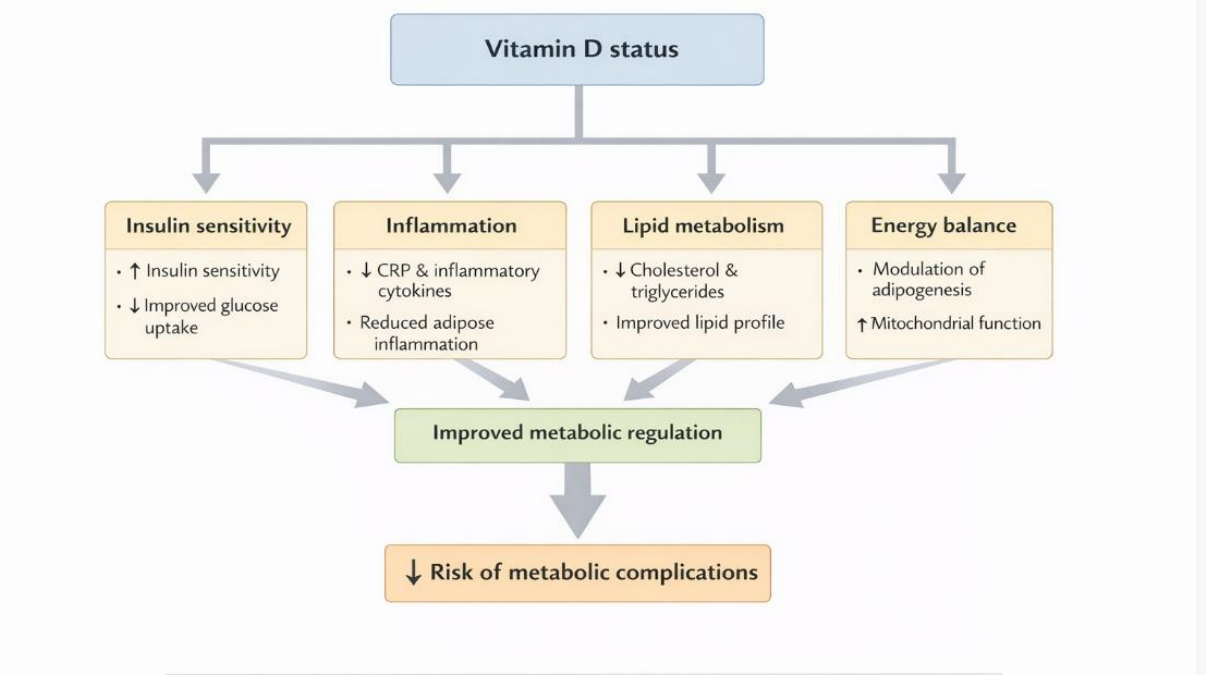

Figure 1. Proposed mechanisms linking vitamin D status to metabolic complications in obesity

The aim of this review is to assess current evidence regarding the effects of vitamin D supplementation on obesity and metabolic syndrome.

Review methods

A narrative literature review was conducted to evaluate current evidence regarding the role of vitamin D supplementation in obesity and metabolic syndrome. Electronic searches were performed in the PubMed database and supplemented with official publications from the World Health Organization, reports from the European Commission, and relevant peer-reviewed literature.

The search strategy used combinations of keywords including “vitamin D,” “vitamin D supplementation,” “obesity,” “overweight,” “metabolic syndrome,” and “insulin resistance.” Studies published in English between 2010 and 2024 were considered. Preference was given to systematic reviews, meta-analyses, randomized controlled trials, and high-quality observational studies evaluating the effects of vitamin D supplementation in overweight or obese populations.

Reference lists of selected publications were also screened to identify additional relevant studies.

Results

Effects on selected metabolic complications in obesity

Effects on insulin resistance.

Clinical trials examining vitamin D (VD) supplementation in healthy individuals, those with impaired glucose tolerance, and patients with type 2 diabetes have produced inconsistent findings regarding its effects on insulin resistance. While observational data suggest a link between low vitamin D status and impaired metabolic function, interventional studies show

variable outcomes (Wamberg et al. 2015). A limited number of small trials showed improved insulin resistance in small subgroups including subjects with VD deficiency and obese men. Findings suggest that vitamin D repletion may be an effective, independent modulator of insulin resistance in vitamin D–deficient populations at high metabolic risk. In a randomized, placebo-controlled trial (von Hurst et al. 2010) of insulin-resistant, vitamin D–deficient South Asian women residing in New Zealand, daily vitamin D_3 supplementation produced robust improvements in insulin sensitivity. Supplementation yielded a significant reduction in HOMA1-IR compared with placebo (−0.25 vs. +0.36; $P = 0.03$), alongside increases in HOMA2 %S ($P = 0.01$) and decreases in fasting insulin ($P = 0.02$). Importantly, these effects persisted after adjustment for regression to the mean ($P = 0.003$). However, no significant changes were observed in BMI, fasting glucose, β-cell function, lipid parameters, or inflammatory markers.

However, a recent meta-analysis (Pramono et al. 2020) of eighteen randomized controlled trials comparing changes in insulin sensivity between VD supplementation and placebo groups revealed no statistically significant overall effect of VD on peripheral insulin sensitivity. The pooled standardized mean difference in change from baseline was −0.01 (95% confidence interval −0.12 to 0.10; $P = 0.87$), with negligible heterogeneity across studies ($I^2 = 0\%$; $P = 0.60$), indicating consistent findings among trials, providing no evidence that VD supplementation has positive results in peripheral insulin sensitivity.

Effects on dyslipidemia

In addition to glucose metabolism, several studies have evaluated the effects of vitamin D supplementation on lipid parameters. A systematic review and meta-analysis (Dibaba 2019) evaluated the effects of vitamin D supplementation on serum lipid profiles across randomized controlled trials. The pooled analysis demonstrated that vitamin D supplementation was associated with significant reductions in total cholesterol, low-density lipoprotein cholesterol, and triglyceride levels, whereas no significant effect was observed for high-density lipoprotein cholesterol. The authors concluded that vitamin D supplementation may provide modest lipid-lowering benefits, particularly in individuals with vitamin D insufficiency, although the clinical relevance of these changes remains to be fully established. However, a more recent meta-analysis (Lu et al. 2024) evaluated the effects of vitamin D supplementation on lipid parameters across randomized controlled trials. Lipid parameters typically assessed in dyslipidemia

include total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). The pooled results demonstrated no significant changes in total cholesterol or LDL-C compared with control groups. Although modest improvements in triglycerides and HDL-C were observed in selected subgroups, the overall findings did not support a consistent lipid-modifying effect of vitamin D supplementation. These results suggest that vitamin D is unlikely to exert clinically meaningful benefits on dyslipidemia when used as a standalone intervention.

Effects on hypertension

A recent systematic review and meta-analysis (Zhang et al. 2020) evaluated the effects of vitamin D supplementation on blood pressure and hypertension in the general population. The analysis of randomized controlled trials demonstrated that, although supplementation effectively increases serum 25-hydroxyvitamin D concentrations, it does not produce significant reductions in either systolic or diastolic blood pressure compared with placebo. The authors concluded that vitamin D supplementation should not be recommended as an antihypertensive intervention at the population level. However, they emphasized the need for further well-designed trials focusing on individuals with vitamin D deficiency and longer intervention periods to clarify potential subgroup benefits.

Effects on inflammatory biomarkers.

In one randomized controlled trial (Shab-Bidar et al. 2012) involving subjects with type 2 diabetes, daily consumption of a vitamin D_3-fortified yoghurt drink (doogh) significantly improved systemic inflammatory profiles relative to a non-fortified control. Participants receiving the fortified beverage exhibited elevated serum 25(OH)D levels accompanied by marked reduction in pro-inflammatory biomarkers, including tumour necrosis factor-α (TNF-α, $p = 0.044$), interleukin-6 (IL-6, $p = 0.002$), highly sensitive C-reactive protein (hsCRP, $p < 0.001$), and serum amyloid A (SAA, $p = 0.022$), alongside an increase in the anti-inflammatory cytokine IL-10 ($p = 0.013$). The between-group differences in changes for hsCRP, SAA, and IL-6 remained statistically significant after adjustment for changes in insulin sensitivity, underscoring the independent effect of improved vitamin D status on inflammatory markers. Another study, a bidirectional Mendelian randomization model (Zhou and Hyppönen 2023), investigated the causal relationship between serum 25-hydroxyvitamin

D [25(OH)D] concentrations and systemic inflammation measured by C-reactive protein (CRP). The analysis demonstrated a non-linear inverse association, whereby increases in genetically predicted vitamin D levels were associated with lower CRP concentrations primarily within the vitamin D deficiency range, with the effect plateauing at higher concentrations. Importantly, the study did not support a causal effect of CRP on vitamin D status, suggesting directionality from vitamin D deficiency toward inflammation. These findings provide evidence that correction of vitamin D deficiency may contribute to reductions in low-grade systemic inflammation, although benefits appear limited to deficient individuals.

Effects on weight management

The randomized controlled trial (Salehpour et al. 2012) demonstrated that vitamin D_3 supplementation (25,000 IU/week for 12 weeks) in overweight and obese women resulted in modest reductions in body fat mass and waist circumference without meaningful weight loss. Importantly, the intervention significantly increased serum 25-hydroxyvitamin D [25(OH)D] concentrations and reduced parathyroid hormone levels, indicating effective correction of vitamin D status. However, changes in body weight and anthropometric indices were not statistically significant between the vitamin D and placebo groups. Furthermore, the authors reported a significant inverse correlation between changes in serum 25(OH)D and body fat mass, suggesting a potential relationship between improved vitamin D status and adiposity regulation.

A randomized controlled trial (Mason et al. 2016) evaluated the effects of vitamin D_3 supplementation during a structured weight-loss program in overweight and obese women. The double-blind, placebo-controlled study demonstrated that supplementation effectively increased serum 25-hydroxyvitamin D [25(OH)D] concentrations, confirming good biochemical response and adherence. However, despite improved vitamin D status, no significant differences were observed between the vitamin D and placebo groups in terms of lean mass preservation, muscle strength, or overall body composition during the weight-loss intervention. These findings suggest that vitamin D supplementation alone does not enhance body composition outcomes in the context of caloric restriction and lifestyle modification, supporting the view that its role in weight management is likely supportive rather than primary. Another recent study, a systematic review and meta-analysis (Perna 2019), which included 11 randomized controlled trials involving 947 participants, evaluated the impact of

vitamin D supplementation on anthropometric outcomes in overweight and obese individuals. The pooled analysis demonstrated a small but statistically significant reduction in body mass index and waist circumference; however, no significant effect on overall body weight was observed. These findings suggest that although vitamin D supplementation may exert modest favorable effects on selected anthropometric parameters, its clinical utility as an independent weight-loss strategy remains limited.

Safety

Vitamin D supplementation is generally safe and well tolerated when used within recommended doses. Most randomized controlled trials report a low incidence of adverse events, with toxicity being rare at typical maintenance doses. However, excessive intake may lead to hypervitaminosis D, characterized primarily by hypercalcemia, hypercalciuria, nephrolithiasis, and, in severe cases, renal impairment. The risk of toxicity is most commonly associated with prolonged high-dose supplementation without biochemical monitoring. Current guidelines therefore emphasize individualized dosing and periodic assessment of serum 25-hydroxyvitamin D and calcium levels, particularly in high-risk populations. Overall, when administered appropriately, vitamin D supplementation demonstrates a favorable safety profile in both general and obese populations.

Discussion

Vitamin D supplementation in individuals with overweight and obesity has been extensively investigated, yet evidence for consistent metabolic benefits remains limited. Observationally, low serum 25-hydroxyvitamin D (25[OH]D) is more prevalent in obesity and is associated with adverse metabolic profiles, including insulin resistance and dysglycemia, likely due to sequestration of fat-soluble vitamin D in adipose tissue and altered metabolism (de Oliveira et al. 2020). Systematic reviews and meta-analyses across RCTs generally show no robust effect of vitamin D supplementation alone on insulin resistance, glycemic indices, or anthropometric markers in overweight and obese populations, although subgroup analyses suggest potential modest benefits under specific conditions (e.g., in youth at higher doses or longer durations) (Pramono et al. 2020). Taken together, current evidence suggests that vitamin D supplementation plays a supportive rather than primary role in the management of obesity-related metabolic disturbances

Conclusions

Current evidence indicates that vitamin D supplementation effectively restores serum 25-hydroxyvitamin D concentrations in individuals with overweight and obesity, but its impact on obesity-related metabolic outcomes remains modest and inconsistent. Across randomized controlled trials and meta-analyses, supplementation alone does not appear to produce clinically meaningful reductions in body weight, body mass index, or blood pressure. Similarly, effects on insulin resistance and glycemic control are heterogeneous, with most pooled analyses demonstrating no significant overall benefit despite occasional positive findings in small or vitamin D–deficient subgroups.

With respect to lipid metabolism and inflammatory status, some studies report favorable changes, including reductions in selected lipid fractions and pro-inflammatory biomarkers. Nevertheless, these effects are generally small, not consistently replicated, and of uncertain clinical relevance. Evidence from weight-management trials further suggests that vitamin D supplementation does not substantially enhance body composition or weight-loss outcomes when added to lifestyle interventions, although modest reductions in fat mass or waist circumference have been observed in certain populations.

Importantly, several sources of heterogeneity likely contribute to the variability of findings, including differences in baseline vitamin D status, dosing regimens, duration of supplementation, degree of adiposity, and co-interventions such as caloric restriction or physical activity. Notably, individuals with documented vitamin D deficiency may derive greater metabolic benefit, supporting a more individualized rather than universal supplementation strategy. From a safety perspective, vitamin D supplementation is generally well tolerated when administered within recommended dosing ranges and with appropriate biochemical monitoring.

In summary, current evidence does not support vitamin D supplementation as an effective standalone therapy for the management of obesity or its major metabolic complications. Its clinical utility appears primarily adjunctive, particularly in populations with confirmed deficiency. Future large, well-designed randomized controlled trials employing standardized dosing strategies, adequate duration of follow-up, and careful stratification by baseline vitamin D status are warranted to clarify potential subgroup-specific benefits and to better define the role of vitamin D in comprehensive obesity management. Future research should

focus on large, well-designed randomized trials with standardized dosing strategies and careful stratification by baseline vitamin D status.

Summary of Selected Studies on Vitamin D Supplementation in Obesity and Metabolic Outcomes

Study (year)	Design	Population	Intervention	Duration	Main outcomes
Salehpour et al., 2012	RCT, double-blind	Overweight/obese women (n=77)	Vitamin D3 25,000 IU/week vs placebo	12 weeks	↓ fat mass ↓waist circumference no significant change in body weight
Mason et al., 2014	RCT, double-blind	Overweight/obese women in weight-loss program	Vitamin D3 vs placebo	12 months	↑ serum 25(OH)D; no significant effect on lean mass or body composition
Perna et al., 2019	Systematic review & meta-analysis	11 RCTs (n=947)	Vitamin D supplementation	Variable	Small ↓ BMI and waist circumference; no significant effect on body weight
Zhang et al., 2020	Systematic review & meta-analysis	General population RCTs	Vitamin D supplementation	Variable	No significant reduction in systolic or

					diastolic blood pressure
Dibaba, 2019	Systematic review & meta-analysis	RCTs on lipid profile	Vitamin D supplementation	Variable	↓ TC, ↓ LDL-C, ↓ TG; no significant effect on HDL-C
Lu et al., 2024	Systematic review & meta-analysis	RCTs on lipid parameters	Vitamin D supplementation	Variable	No consistent effect on TC or LDL-C; minor subgroup effects only

Table2 . Summary of Selected Studies on Vitamin D Supplementation in Obesity and Metabolic Outcomes

Disclosure

The authors declare that they have no relevant financial or non-financial interests to disclose

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