

Vitamin D and Cardiovascular Risk in Obese Children in Saudi Arabia: A Narrative Review

Abdullah Ibrahim Al-Sharif¹, Tasneem Rashed Adam², Zainab Ali Alaraibi³, Ruqayah Abbas Abdulkarem Ahmed⁴, Baland Mohammed Kordi⁵, Faisal Aijaz Amin Shah⁶, Mohammed Wadia Abumadian⁷, Ibrahim Taha I. AlFajri⁸, Amal Baalqasim Altayeb Sulaimani⁹, Mohamed Ali Mohammed Homoq¹⁰, Mohamed Abdullatif Al-Dajani¹¹

¹Department of Business, College of Medicine, Alfaisal University, Riyadh, 11554, Saudi Arabia

²Dental Health Department, College of Applied Medical Sciences, King Saud University, Riyadh, 11451, Saudi Arabia

³ICU, King Khalid Hospital, Najran, Saudi Arabia

⁴Department of Rheumatology, Saudi German Hospital, Dubai, United Arab Emirates

⁵Family Medicine, King Abdulaziz University Hospital, 44321, Saudi Arabia

⁶Internal Medicine, Security Forces Hospital, Makkah, 24235, Saudi Arabia

⁷Plastic and Reconstructive Surgery, King Fahad Armed Forces Hospital, Jeddah, 23523, Saudi Arabia

⁸Emergency Department, Al Jabr Eye and ENT Hospital, Al-Ahsa, 36342, Saudi Arabia

⁹Radiology, King Fahad Hospital, 42386, Saudi Arabia

¹⁰College of Medicine, University Sains Malaysia, George Town, 16150, Malaysia

¹¹Family Medicine Department, Primary Health Care, Dammam, 32241, Saudi Arabia

Author Designation: ¹Assistant Professor, ²Researcher, ³Resident, ⁴Clinical Attachment, ^{5,10}Medical Intern, ⁶R1 Resident, ^{7,8}General Practitioner, ⁹Radiology Resident, ¹¹Medicine Resident

*Corresponding author: Tasneem Rashed Adam (e-mail: tasneemr.m94@gmail.com).

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Abstract Background: Childhood obesity and vitamin D deficiency are highly prevalent in Saudi Arabia and increasingly recognized as interconnected risk factors for cardiometabolic complications. Their frequent coexistence may amplify cardiovascular risk from an early age. This review aims to summarize the prevalence of vitamin D deficiency among obese children in Saudi Arabia, examine the mechanistic links to cardiovascular risk factors and situate regional findings within the context of global evidence. **Methods:** A narrative review was conducted of studies published between 2000 and 2025 using PubMed, Google Scholar and the Saudi Digital Library. Eligible studies included cross-sectional, cohort, case-control and clinical trials that evaluated vitamin D status in relation to cardiovascular risk markers among children under 18 years of age. **Findings:** Available evidence demonstrates that vitamin D deficiency in obese children is associated with dyslipidaemia, insulin resistance, elevated blood pressure, vascular dysfunction and early manifestations of atherosclerosis. In Saudi Arabia, deficiency rates frequently exceed 80% despite abundant sunlight, largely due to cultural clothing practices, limited outdoor activity and inadequate dietary intake. Local studies also highlight early indications of myocardial dysfunction and metabolic impairment among vitamin D-deficient youth. However, most investigations are cross-sectional, include small sample sizes and often fail to adjust for confounders such as diet and sun exposure. **Conclusion:** The combined burden of obesity and vitamin D deficiency in Saudi children constitutes a pressing public health concern with significant long-term cardiovascular implications. Policy measures such as incorporating vitamin D screening into obesity management, nationwide supplementation programs and structured health education campaigns are urgently needed. Future research should emphasize large-scale prospective cohorts, randomized controlled trials and systematic reviews to establish causal pathways and inform evidence-based interventions.

Key Words Vitamin D, Childhood Obesity, Cardiovascular Risk, Saudi Arabia, Paediatrics, Cardiometabolic Health

INTRODUCTION

Childhood obesity and vitamin D deficiency are increasingly recognized as critical public health concerns worldwide, including in Saudi Arabia. The global rise in paediatric obesity has been paralleled by a surge in metabolic and cardiovascular complications [1]. In Saudi Arabia, this burden is compounded by sedentary lifestyles and unhealthy dietary habits. Despite the country's year-round sunlight, vitamin D deficiency remains alarmingly common among

children and adolescents, largely due to limited sun exposure and inadequate dietary intake [2]. Both obesity and hypovitaminosis D have been independently linked to cardiometabolic risk factors such as insulin resistance and hypertension [1,3]. Importantly, early indicators of cardiovascular disease, such as increased carotid intima-media thickness, have already been reported in obese children with vitamin D deficiency, suggesting that atherosclerotic changes may begin as early as childhood [4].

Vitamin D, a fat-soluble prohormone, plays a central role in calcium balance, bone metabolism and immune regulation. It is synthesized cutaneous through ultraviolet B radiation and obtained from dietary sources or supplementation [8,6]. Once produced, vitamin D is metabolized in the liver to 25-hydroxyvitamin D [25(OH)D], the major circulating form and the most reliable marker of vitamin D status. It is subsequently hydroxylated in the kidneys to form 1,25-dihydroxyvitamin D (calcitriol), the biologically active form that exerts effects on multiple tissues by binding to the vitamin D receptor and regulating gene expression [7].

Extensive research has demonstrated strong associations between childhood obesity and cardiometabolic disturbances, including elevated blood pressure, dyslipidaemia and vascular changes such as increased carotid intima-media thickness [8,9]. Vitamin D deficiency has emerged as a potentially modifiable factor in this context. Low serum 25(OH)D concentrations have been linked to adverse lipid patterns, including higher triglycerides and non-HDL cholesterol, alongside reduced HDL cholesterol, alterations that contribute to increased atherogenic risk in obese children and adolescents [8,10].

Although many observational studies have investigated associations between vitamin D deficiency and cardiovascular risk, results remain inconsistent. Moreover, relatively few reviews have focused on paediatric populations within Saudi Arabia, where the coexistence of obesity and hypovitaminosis D presents a particularly urgent challenge. To address this gap, the present narrative review aims to: (1) summarize prevalence data on vitamin D deficiency among obese children reported in studies from 2000 to 2025; (2) explore mechanistic pathways linking low vitamin D status to cardiometabolic risk factors, including dyslipidaemia, insulin resistance (HOMA-IR), hypertension, vascular dysfunction such as carotid intima-media thickening and inflammation; and (3) compare findings from Saudi Arabia with international evidence to highlight their clinical and public health implications. Accordingly, this review is structured to present prevalence data, discuss mechanistic associations and outcomes, contrast Saudi and global findings and conclude with implications for prevention and management strategies.

METHODS

This review focused on research examining the relationship between vitamin D status and cardiovascular risk among obese children, with particular emphasis on populations in Saudi Arabia. A narrative literature search was conducted for the period 2000-2025 using PubMed, Google Scholar and the Saudi Digital Library. The search strategy employed the following keywords: “vitamin D,” “obesity,” “children,” “paediatric,” “cardiovascular risk,” “Saudi Arabia,” and “Gulf countries.”

Studies were eligible if they were published in English, involved human participants under the age of 18 and explored vitamin D status in relation to cardiovascular outcomes. Both regional and international studies were

included, covering cross-sectional, cohort, case-control and clinical trials, to provide a broader global context alongside local evidence. Animal studies, case reports and studies not assessing cardiovascular outcomes were excluded.

Data extraction was carried out independently by two reviewers using a structured framework. Extracted information included study design, sample size, participant characteristics, methods of vitamin D assessment, prevalence of deficiency and reported cardiovascular markers such as lipid profiles, blood pressure, insulin resistance, carotid intima-media thickness and myocardial function. Discrepancies between the two reviewers were resolved by consultation with a third reviewer.

Mechanisms Linking Obesity to Reduced Vitamin D Bioavailability in Children

Although vitamin D is classically recognized for its role in calcium balance and bone health, it is increasingly understood to exert wider physiological effects, including regulation of immune function, insulin sensitivity, inflammation and cardiovascular health [11]. These functions are particularly important in paediatric obesity, a condition often accompanied by metabolic dysregulation. A consistently high prevalence of vitamin D deficiency has been reported among obese children, with several physiological and behavioural mechanisms proposed to explain this relationship.

One key mechanism is the sequestration of vitamin D within adipose tissue. As a fat-soluble compound, vitamin D is stored in excess adipose mass, leading to reduced circulating levels of 25-hydroxyvitamin D despite normal or elevated body stores [12]. This sequestration reduces the bioavailable fraction of vitamin D needed for metabolic and cardiovascular functions. Beyond physiology, lifestyle factors further exacerbate the problem. Obese children are more likely to engage in sedentary behaviours, spend longer periods indoors and have limited exposure to sunlight, reducing cutaneous synthesis of vitamin D [13]. Poor dietary intake, characterized by inadequate consumption of vitamin D-rich foods or fortified products, further contributes to deficiency.

In Saudi Arabia, these risk factors are compounded by sociocultural and environmental conditions. Despite abundant sunlight, vitamin D deficiency is highly prevalent across all age groups, including children and adolescents. Cultural clothing practices that limit skin exposure, along with extreme heat that discourages outdoor activity, significantly restrict opportunities for vitamin D synthesis. Additionally, dietary intake of fortified foods and oily fish remains insufficient. Wimalawansa [14] reported that hypovitaminosis D is particularly widespread across Middle Eastern populations, especially among women. National and regional studies within Saudi Arabia indicate deficiency rates exceeding 80%, with some cohorts reporting levels as high as 90%. These figures are particularly concerning when considered alongside escalating rates of childhood obesity, which magnify the risk of vitamin D deficiency and its downstream metabolic and cardiovascular consequences.

Early Cardiovascular Alterations Associated with Childhood Obesity

Childhood obesity has become a growing global health challenge, affecting both developed and developing nations [15]. In 2010, Asia recorded the highest absolute number of overweight and obese children, largely due to its large population, even though the prevalence was relatively low at 4.9%. In comparison, Africa showed a higher prevalence of 8.5%, projected to increase to 12.7% by 2020. At a global level, the prevalence of childhood overweight and obesity rose from 4.2% in 1990 to 6.7% in 2010 and was expected to reach 9.1% by 2020 [16].

Beyond prevalence, the clinical implications are profound. Obese children face an elevated risk of cardiovascular complications, many of which begin in childhood and track into adulthood. Atherosclerosis, once considered a disease of later life, has been shown to originate in the first decade of life, particularly among children with obesity and comorbid conditions such as dyslipidaemia, hypertension and insulin resistance [17]. Longitudinal data further demonstrate that elevated childhood low-density lipoprotein cholesterol (LDL-C) is linked with increased carotid intima-media thickness and impaired vascular function in adulthood [17].

The clustering of risk factors is also evident at an early age. A study of obese children aged 10-12 years reported that nearly 13% met the criteria for metabolic syndrome, with elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C) and high blood pressure as key contributors to cardiovascular risk [18]. Moreover, obese

children with metabolic syndrome often exhibit elevated high-sensitivity C-reactive protein (hs-CRP) levels, an inflammatory biomarker associated with early cardiovascular disease [19].

Interpretation of findings across studies is complicated by methodological differences, including study design (cross-sectional versus longitudinal), definitions of vitamin D deficiency (<20 ng/mL versus <30 ng/mL) and the choice of outcome measures (e.g., lipid abnormalities, vascular changes, inflammatory markers). Interestingly, cohorts from Saudi Arabia often report higher prevalence rates compared with international studies, reflecting not only contextual differences but also variations in methodology.

Vitamin D Deficiency as a Mediator of Inflammation, Insulin Resistance and Vascular Dysfunction

A growing body of evidence links vitamin D deficiency with increased cardiovascular risk in children, particularly in those with obesity. In one study of 178 overweight and obese paediatric patients, children with serum 25-hydroxyvitamin D [25(OH)D] concentrations below 20 ng/mL exhibited significantly higher levels of non-HDL cholesterol, triglycerides and atherogenic lipid ratios such as TG/HDL and TC/HDL, markers strongly predictive of cardiovascular disease [8]. Similarly, a case-control study of obese prepubertal children found that vitamin D deficiency was associated with significantly higher diastolic blood pressure percentiles and demonstrated a positive correlation with HDL levels, suggesting early vascular alterations [20].

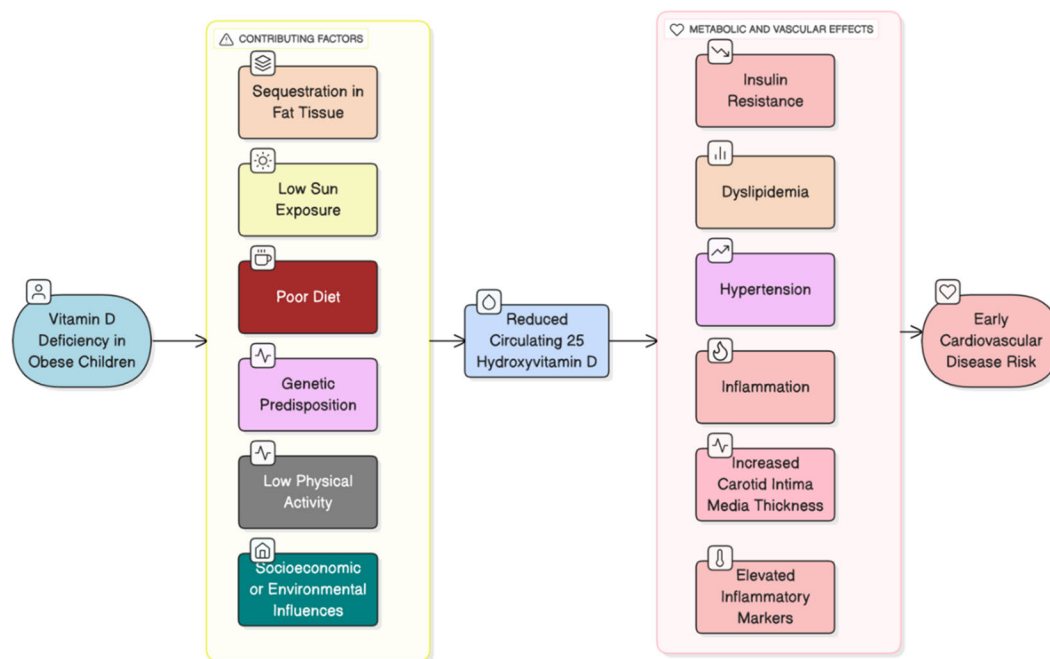


Figure 1: Conceptual framework illustrating the pathways linking vitamin D deficiency in obese children to early cardiovascular disease risk. Contributing factors (left) reduce circulating 25-hydroxyvitamin D, which in turn mediates metabolic and vascular effects (right), culminating in increased early cardiovascular risk

Several biological mechanisms help explain these associations. Vitamin D plays a role in regulating inflammatory pathways, endothelial function and insulin sensitivity, all of which are key determinants of cardiovascular health. As illustrated in Figure 1, factors contributing to vitamin D deficiency in obese children, such as adipose tissue sequestration, low dietary intake and reduced sun exposure, lead to decreased circulating 25(OH)D, which in turn exacerbates metabolic disturbances and vascular dysfunction. Deficiency also promotes vascular stiffness and calcification, both of which are central processes in the pathogenesis of atherosclerosis [21].

Evidence from a retrospective study of 376 children with severe obesity showed that lower 25(OH)D levels were significantly associated with reduced HDL-C concentrations and a twofold greater risk of low HDL, an established cardiovascular risk marker [10]. Insulin resistance, another key driver of cardiovascular disease, is also worsened by hypovitaminosis D. For instance, in a cohort of 310 obese children, vitamin D deficiency was strongly linked with higher HOMA-IR values and atherogenic dyslipidaemia, even after adjusting for body mass index and age [11]. Likewise, a Spanish study involving 120 obese children demonstrated significantly higher triglycerides and insulin resistance among those with vitamin D deficiency compared to those with sufficient levels [22]. Nonetheless, the absence of detailed dietary and sun-exposure data in many studies limit interpretation, as these are important confounders in the relationship between vitamin D and insulin resistance.

Finally, carotid intima-media thickness (c-IMT), a well-established subclinical marker of atherosclerosis, has also been associated with vitamin D status. In a clinical study of 247 obese children, low vitamin D levels were significantly correlated with increased c-IMT and a higher prevalence of metabolic syndrome, suggesting structural vascular changes that predispose children to future cardiovascular disease [3].

High Prevalence of Deficiency and Cardiovascular Risk Markers in Saudi Youth

Vitamin D deficiency remains a pervasive and persistent public health challenge among Saudi children, with numerous studies reporting alarmingly high prevalence rates across regions and age groups. A cross-sectional study conducted in Jeddah demonstrated that 97.5% of children aged 2-18 years were either deficient or insufficient, with only 2.5% maintaining normal vitamin D levels [23]. Nationwide data from Kaddam *et al.* further highlighted the scope of the problem, showing deficiency in 49.5% of schoolchildren, with higher rates among females, obese students and children living in the Eastern Region. Although this study employed robust anthropometric and biochemical assessments, its reliance on self-reported lifestyle and dietary data raised concerns about reporting bias. Additionally, potential confounders such as smoking and detailed dietary patterns were not fully considered, which may partly explain variations in vitamin D status [24].

Regional data confirm similar trends. A hospital-based study in Riyadh found vitamin D deficiency in 17.6% and insufficiency in 25.6% of children, with older children (10-14 years) being the most affected group [25]. In the Aseer region, nearly one-quarter (24.6%) of children under two years were deficient and 45.9% insufficient. Exclusive breastfeeding, limited sun exposure and urban living were identified as major contributors [26]. A broader review of national data between 2011 and 2016 concluded that, on average, 81% of Saudi children and adolescents were vitamin D deficient, underscoring the chronic and widespread nature of the problem despite the country's ample sunlight [27].

The high prevalence of vitamin D deficiency is particularly concerning in the context of obesity, given its association with cardiometabolic alterations. Studies among Saudi youth have demonstrated that low vitamin D levels are linked with early cardiovascular dysfunction. For example, research in adolescents found associations between deficiency and subclinical myocardial impairment, including elevated left and right ventricular Tei indices and reduced systolic velocities, suggesting early cardiac dysfunction [28]. A systematic review also confirmed that vitamin D deficiency increases the risk of type 2 diabetes and worsens glycaemic control, largely mediated by insulin resistance [29].

Taken together, these studies highlight two converging themes: (1) vitamin D deficiency is highly prevalent among Saudi children and adolescents and (2) it is consistently associated with early markers of cardiometabolic dysfunction. Some investigations emphasize structural outcomes, such as myocardial dysfunction [28], whereas others highlight metabolic disturbances, including insulin resistance and impaired glycaemic control [29]. Despite variability in methods and outcomes, the evidence collectively supports a multifactorial, synergistic role for vitamin D deficiency in amplifying cardiovascular risk in obese youth.

Although paediatric studies remain limited, evidence from Saudi adult cohorts provides additional context regarding the broader cardiometabolic consequences of vitamin D deficiency. In young Saudi women, low vitamin D status was inversely associated with waist circumference, blood pressure and parathyroid hormone levels, with those in the lowest vitamin D group exhibiting the highest burden of cardiometabolic risk factors [30]. Among patients with coronary heart disease, vitamin D deficiency was significantly associated with diabetes, although not with cholesterol levels or hypertension [31]. A large community-based study in Riyadh reported that deficiency was linked to sex-specific lipid abnormalities, low HDL in men and elevated triglycerides in women [32]. Furthermore, obese Saudi adults with type 2 diabetes exhibited lower vitamin D levels alongside higher levels of inflammatory cytokines (IL-6, TNF- α and CRP) and greater insulin resistance, pointing to inflammation as a key pathway linking deficiency to cardiovascular risk [33].

International adult studies offer further mechanistic insights and long-term outcome data that help contextualize findings in children. In the prospective Framingham Offspring Study, which followed 1,739 middle-aged adults without baseline cardiovascular disease for a mean of 5.4 years, those with serum 25-hydroxyvitamin D levels below 15 ng/mL had a 62% greater risk of developing a first major cardiovascular event, with risk more than doubling among participants with hypertension [34]. In contrast, a systematic review and meta-regression by Nudy *et al.* (2020) synthesizing 22 randomized controlled trials (RCTs) with over 83,000 participants found no significant reduction in cardiovascular outcomes, including myocardial infarction, stroke or cardiovascular mortality, following vitamin D supplementation. Importantly, baseline vitamin D status did not modify the results, suggesting that supplementation alone may not provide cardiovascular protection [35].

Several explanations may account for these discrepancies. Most RCTs were not specifically designed or powered to detect cardiovascular endpoints, supplementation regimens varied considerably in dose and duration and many participants were not vitamin D deficient at baseline. In addition, biological factors such as obesity-related sequestration of vitamin D in adipose tissue and the absence of critical co-nutrients (e.g., magnesium, vitamins A and K) necessary for optimal vitamin D metabolism and function may have attenuated the observed effects [36,37].

In summary, while observational studies consistently demonstrate strong associations between low vitamin D levels and adverse cardiometabolic outcomes [30-34,37], evidence from large RCTs remains inconclusive [35]. These conflicting findings underscore the complexity of vitamin D's role in cardiometabolic health and highlight the need for rigorously designed, targeted intervention studies in high-risk populations, particularly obese children and adolescents in regions like Saudi Arabia where deficiency is widespread.

Policy Gaps and Opportunities for Vitamin D Supplementation in Saudi Arabia

Early detection of vitamin D deficiency in obese children is particularly important in Saudi Arabia, where both conditions are highly prevalent and strongly linked to cardiometabolic complications. Screening provides an opportunity to intervene before the onset of chronic diseases, as deficiency has been associated with insulin resistance, inflammation, impaired bone health and even subtle myocardial dysfunction in adolescents [28,38]. These findings highlight the need for early cardiovascular risk assessment alongside obesity management.

To address widespread deficiency, the Saudi Food and Drug Authority (SFDA) has established regulatory guidelines defining safe intake thresholds, with an upper limit of 25 µg/day for children and adolescents [39]. While these benchmarks form the foundation for national strategies, real-world outcomes suggest limited success. For instance, a systematic review of studies conducted between 2012 and 2023 found that approximately 81% of Saudi

children and adolescents had suboptimal vitamin D levels [40]. Similarly, a cross-sectional study in Majmaah reported that over 94% of children aged 0-12 years were either deficient or insufficient [14]. These alarming statistics underscore a clear gap between policy recommendations and public health outcomes, suggesting that current interventions are insufficient without stronger implementation at the population level. Integrating SFDA guidelines into both clinical practice and national health programs may help bridge this gap, particularly for high-risk groups such as obese children.

Correcting vitamin D deficiency has the potential to reduce cardiovascular risk factors, including hypertension, dyslipidaemia and insulin resistance [41]. Supplementation is especially important in obese children, whose reduced sun exposure and lifestyle habits make endogenous synthesis less reliable [23]. Evidence from national datasets further shows that supplement use is associated with significantly lower rates of deficiency, supporting the value of structured supplementation programs [42].

A coordinated national strategy is therefore urgently needed, one that incorporates vitamin D screening, supplementation and dietary guidance into childhood obesity prevention and treatment programs [24]. A systematic review emphasized the importance of locally tailored guidelines to identify at-risk groups, standardize diagnostic criteria and harmonize treatment protocols across healthcare systems [27].

Emerging research also points to novel biological mechanisms linking vitamin D status with adipose tissue metabolism. In a one-year lifestyle intervention study in Québec, Canada, Gangloff *et al.* reported that increases in serum 25(OH)D were independently associated with decreases in leptin levels, even after adjusting for adiposity. A 27% rise in vitamin D corresponded with a 27% reduction in leptin, suggesting a physiologically meaningful inverse relationship [43]. Similarly, Hengist *et al.* proposed that in individuals with obesity, vitamin D stored in adipose tissue may become functionally unavailable due to impaired lipolysis and adipocyte dysfunction. Their review highlighted that physical activity, independent of weight loss, may mobilize vitamin D from fat stores, thereby improving circulating levels [44].

However, these findings are based on populations from temperate countries such as the United States, Germany, Japan and the United Kingdom, where outdoor activity and sun exposure are relatively common. In contrast, Saudi Arabia presents a unique challenge. Extreme heat, cultural clothing practices and predominantly indoor lifestyles limit opportunities for sun-driven vitamin D synthesis, leading to chronically low levels despite abundant sunlight [14,40,45]. This suggests that unlike in temperate regions, the primary issue in Saudi Arabia is inadequate synthesis rather than sequestration in adipose tissue.

Intervention trials highlight the potential benefits of supplementation in obese and vitamin D-deficient individuals. In Turkey, Bilici *et al.* (2019) demonstrated that

2,000 IU/day of vitamin D administered for three months in obese adolescents reduced BMI standard deviation score, hip circumference, total cholesterol, LDL-C, HbA1c, parathormone and interleukin-6 levels [46]. Although no significant changes were observed in insulin resistance indices such as fasting glucose or HOMA-IR, the improvements in lipid and inflammatory markers are clinically meaningful. Similarly, Imga *et al.* (2018) found that six months of vitamin D₃ supplementation in overweight and obese premenopausal women with severe deficiency improved LDL-C levels and insulin sensitivity [47]. In another study of 376 obese children in the United States, Iqbal *et al.* reported a positive correlation between serum 25(OH)D and HDL-C, further supporting its potential cardioprotective role [10]. However, the omission of key confounders such as diet, pubertal stage and physical activity remains a limitation.

While findings vary across studies, the evidence as a whole supports the safety of supplementation and its potential to improve cardiometabolic outcomes, particularly in populations with severe baseline deficiency [10,46,47]. Although effects on insulin resistance and lipid profiles may be modest or inconsistent, the broader benefits, including improved bone health, inflammatory regulation and potential cardiovascular protection, make vitamin D supplementation a critical public health intervention.

From a population health perspective, tackling vitamin D deficiency in Saudi children should be a national priority. Coordinated strategies, including mandatory fortification, supplementation programs and school-based health education, could substantially reduce long-term cardiometabolic risks.

CONCLUSIONS

The dual burden of obesity and vitamin D deficiency among Saudi children represents a critical and growing public health challenge with significant long-term cardiovascular implications. Current evidence consistently links low vitamin D status with cardiometabolic disturbances, including dyslipidaemia, insulin resistance, vascular dysfunction and early atherosclerotic changes. Yet, the majority of studies are cross-sectional, rely on relatively small cohorts and often overlook important confounders such as sun exposure, diet and pubertal stage, factors that limit causal interpretation and contribute to variability in findings.

Mechanistic insights suggest that vitamin D plays a pivotal role in modulating inflammation, insulin sensitivity and endothelial function. In the context of paediatric obesity, deficiency appears to exacerbate metabolic stress, thereby accelerating cardiovascular risk from an early age. These pathways highlight the urgency of addressing both conditions simultaneously through public health strategies that promote healthier lifestyles, balanced nutrition and adequate vitamin D intake.

At the policy level, incorporating vitamin D screening into national health strategies, particularly with targeted

protocols for high-risk groups, would enable earlier detection and timely management. Structured awareness campaigns aimed at correcting misconceptions, improving dietary practices and encouraging safe sun exposure are particularly important for vulnerable groups such as adolescent females, children with limited outdoor activity and those with severe obesity or metabolic syndrome.

Future research in Saudi Arabia should prioritize prospective cohort studies and randomized controlled trials that employ standardized definitions of vitamin D deficiency, integrate detailed lifestyle assessments and evaluate clinically meaningful cardiovascular outcomes such as carotid intima-media thickness, myocardial function and glycaemic control. Additionally, systematic reviews and meta-analyses of regional data could help synthesize existing evidence, refine clinical guidelines and inform effective interventions tailored to the Saudi population.

Limitations

This review has several limitations. First, as a narrative review, it did not apply systematic methods such as meta-analysis, limiting the ability to quantify associations or infer causality. Second, most studies conducted in Saudi Arabia were cross-sectional and based on small hospital- or school-based samples, reducing their generalizability to the wider paediatric population. Third, definitions of vitamin D deficiency varied (<20 ng/mL vs. <30 ng/mL), as did the outcomes assessed (lipid profiles, insulin resistance, carotid intima-media thickness, myocardial function), complicating direct comparisons. Fourth, key confounding variables, such as sun exposure, diet, pubertal stage and skin pigmentation, were often unmeasured, potentially explaining some inconsistencies in findings. Finally, the review was restricted to English-language studies published between 2000 and 2025, which may have excluded additional relevant work published in Arabic or outside this timeframe.

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