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Narrative Review on Vitamin D Deficiency in India: A Silent Public Health Problem

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ABSTRACT

Vitamin D deficiency is among the most common yet least recognised nutritional problems in India. Despite abundant sunshine, studies from across the country report that a large majority of apparently healthy children, adults and pregnant women have serum 25-hydroxyvitamin D [25(OH)D] concentrations below accepted thresholds, with published estimates ranging from about 40% to 99%. This narrative review summarises vitamin D physiology and mechanism of action, the epidemiology of deficiency in India, its determinants, its skeletal and extraskeletal consequences, and the evidence for supplementation and food fortification. Deficiency in India reflects a convergence of limited effective ultraviolet-B exposure, atmospheric pollution, diets that supply little vitamin D and calcium and are rich in phytate, minimal fortification of staple foods, and increased requirements during growth, pregnancy and ageing. The skeletal consequences, namely rickets, osteomalacia and secondary hyperparathyroidism, are well established. In contrast, large randomised trials in largely vitamin D-replete Western populations have not shown benefits for cancer, cardiovascular disease, diabetes, fractures or falls, and these trials say little about severely deficient populations. Representative Indian data are scarce. We argue that India needs a national vitamin D survey with standardised assays and cut-offs, targeted supplementation for high-risk groups, and broader fortification, rather than indiscriminate testing and high-dose supplementation.*Key words*: Amyotrophic lateral sclerosis; motor neuron disease; vitamin E; vitamin D; methylcobalamin; vitamin B_{12}; nicotinamide riboside; carotenoids; antioxidants; oxidative stress.

Key words: Vitamin D; 25-hydroxyvitamin D; India; public health; fortification; rickets; osteomalacia; secondary hyperparathyroidism.

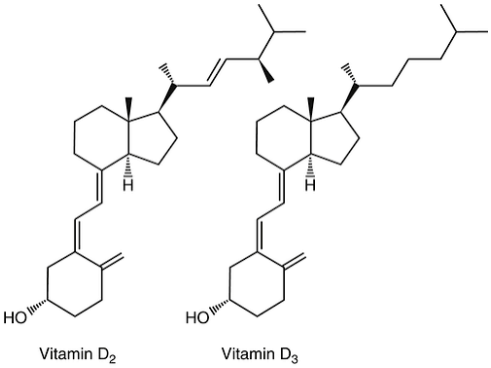
1. Introduction

Vitamin D is unusual among nutrients. It is made in the skin on exposure to sunlight, it behaves as a prohormone, and its active metabolite acts through a nuclear receptor present in many tissues. Vitamin D deficiency is widely described as pandemic, yet it is also regarded as the most under-diagnosed and under-treated nutritional deficiency in the world [1]. India presents a paradox. It is a tropical country with plentiful sunshine, but reviews of the Indian literature report deficiency in 70%–100% of the general population [1], and a more recent review found prevalence between 40% and 99%, with most studies reporting 80%–90% [2]. Deficiency has been documented in all age groups and in urban and rural settings alike [1,2].

The problem is “silent” for several reasons. Subclinical deficiency produces no obvious symptoms, so it is rarely suspected unless frank bone disease appears [1,3]. Vitamin D status is not part of routine public health surveillance, and deficiency is distributed across age, sex and socioeconomic strata, so it does not announce itself as a problem of any one group [1]. At the same time, there is genuine scientific debate about which 25(OH)D concentration is optimal and whether correcting low levels prevents disease beyond the skeleton [4,5]. These uncertainties make it important to separate what is firmly established from what remains hypothesis.

This narrative review describes the physiology and mechanism of action of vitamin D, summarises the epidemiology of deficiency in India, discusses its determinants and consequences, evaluates the evidence from recent trials, and outlines policy options for India.

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2. Vitamin D: Sources, Metabolism and Mechanism of Action

Two forms of the vitamin exist: vitamin D_2 (ergocalciferol), derived from plants and fungi, and vitamin D_3 (cholecalciferol), made in human skin and found in animal foods. In the skin, ultraviolet-B radiation (290–315 nm) converts 7-dehydrocholesterol to previtamin D_3, which isomerises thermally to vitamin D_3. Few foods contain appreciable amounts of vitamin D naturally, and fortified foods and supplements therefore matter [3].

Vitamin D from skin or diet is carried in blood bound to vitamin D-binding protein. It is hydroxylated in the liver to 25(OH)D, the main circulating form and the clinical marker of status, and then in the kidney and other tissues by 1α-hydroxylase (CYP27B1) to 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], the ligand of the vitamin D receptor (VDR). The enzyme CYP24A1 inactivates both 25(OH)D and 1,25$(OH)_2$D [3,4] (Figure 1). The VDR forms a heterodimer with the retinoid X receptor, binds vitamin D response elements and regulates gene transcription. Because the VDR and the vitamin D-metabolising enzymes are expressed widely, vitamin D signalling is thought to have many extraskeletal effects, although the physiological meaning of non-genomic actions is still poorly understood [4].

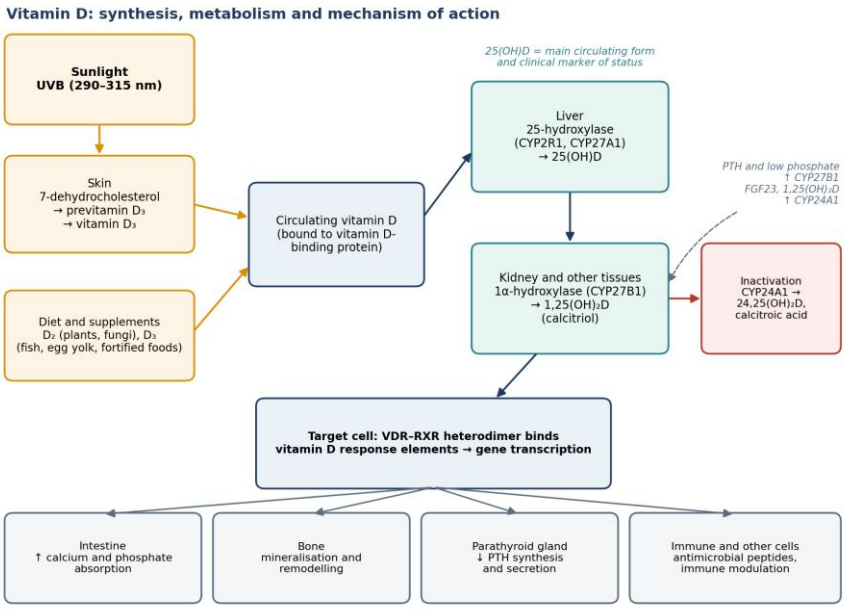


Figure 1. *Synthesis, metabolism and mechanism of action of vitamin D. UVB: ultraviolet B; PTH: parathyroid hormone; FGF23: fibroblast growth factor 23; VDR: vitamin D receptor; RXR: retinoid X receptor. Scheme prepared by the author from refs* [3,4].

The classical actions concern mineral metabolism. 1,25$(OH)_2$D increases intestinal absorption of calcium and phosphate, supports bone mineralisation and suppresses parathyroid hormone (PTH). When vitamin D is lacking, calcium absorption falls, PTH rises (secondary hyperparathyroidism), bone turnover accelerates, and in severe or prolonged deficiency defective mineralisation produces rickets in children and osteomalacia in adults [3,4].

Most authorities define deficiency as a serum 25(OH)D concentration below 20 ng/mL (50 nmol/L) and insufficiency as 21–29 ng/mL (1 ng/mL = 2.5 nmol/L) [3,6]. Indian studies have used different cut-offs: for example, below 20 ng/mL as deficient in a south Indian population study [8] and below 10 ng/mL as "severe" deficiency in a Delhi survey [11]. The 2024 Endocrine Society guideline also notes that no clear evidence defines the optimal target concentration for disease prevention [5]. These differences must be kept in mind when comparing prevalence figures.

3. Burden of Vitamin D Deficiency in India

Early reports from Delhi showed that healthy adults, pregnant women and newborns in almost every group studied had subnormal 25(OH)D concentrations. Only the group with maximal direct sunlight exposure escaped. Deficient groups tended to show disturbed bone mineral homeostasis, with low calcium, raised PTH or biochemical osteomalacia, especially in winter and with low dietary calcium and high dietary phytate [7].

A population study in Andhra Pradesh examined 943 urban and 205 rural healthy adults. Deficiency (below 20 ng/mL) was present in 62% of urban men and 75% of urban women, and in 44% of rural men and 70% of rural women. Mean dietary calcium intake was low relative to Indian recommendations, and the dietary phytate-to-calcium ratio was high, especially in rural subjects [8]. Among 5,137 apparently healthy schoolchildren aged 10–18 years in New Delhi, clinical evidence of vitamin D deficiency was seen in 10.8%, biochemical hypovitaminosis D was highly prevalent, and bone mineral density was lower in children from the lower socioeconomic group [9]. Studies of pregnant women and their newborns in northern India also report a high prevalence of deficiency [10].

A more recent survey in the National Capital Region of Delhi, designed to describe population prevalence, found median 25(OH)D of 7.7 ng/mL in urban and 16.2 ng/mL in rural adults. Severe deficiency (below 10 ng/mL) affected 71% of urban and 20% of rural adults, and deficiency was more frequent in women and in obese individuals [11] (Figure 2). Table 1 summarises these and other studies.

Table 1. Selected studies and reviews of vitamin D status in India.

Study (ref.)	Setting and population	Key findings
Goswami et al., 2000 [7]	Delhi; healthy adult groups, pregnant women and newborns	All groups except the one with maximal direct sunlight exposure had subnormal 25(OH)D; deficient groups showed low calcium, raised PTH or chemical osteomalacia, especially in winter.
Agarwal et al., 2002 [13]	Delhi; toddlers (9–24 months) in high- and low-pollution areas	Mean 25(OH)D 12.4 *vs* 27.1 ng/mL; atmospheric pollution linked to lower status and rickets risk.
Marwaha et al., 2005 [9]	New Delhi; 5,137 schoolchildren aged 10–18 years	Clinical signs of vitamin D deficiency in 10.8%; high prevalence of biochemical hypovitaminosis D; lower bone density in lower socioeconomic group.
Sachan et al., 2005 [10]	Northern India; pregnant women and their newborns	High prevalence of vitamin D deficiency in mothers and newborns.
Harinarayan et al., 2007 [8]	Andhra Pradesh; 943 urban and 205 rural healthy adults	Deficiency (<20 ng/mL) in 62% urban men, 75% urban women, 44% rural men, 70% rural women; low calcium and high phytate intake.
Praveen et al., 2023 [11]	National Capital Region of Delhi; urban and rural adults	Median 25(OH)D 7.7 (urban) and 16.2 (rural) ng/mL; severe deficiency in 71% urban and 20% rural adults; higher in women and obese.
Ritu and Gupta, 2014 [1]	Review of Indian literature	Prevalence of 70%–100% in the general population; all strata affected.
Aparna et al., 2018 [2]	Review of Indian literature	Prevalence 40%–99%, most studies 80%–90%; all age groups and high-risk groups affected.

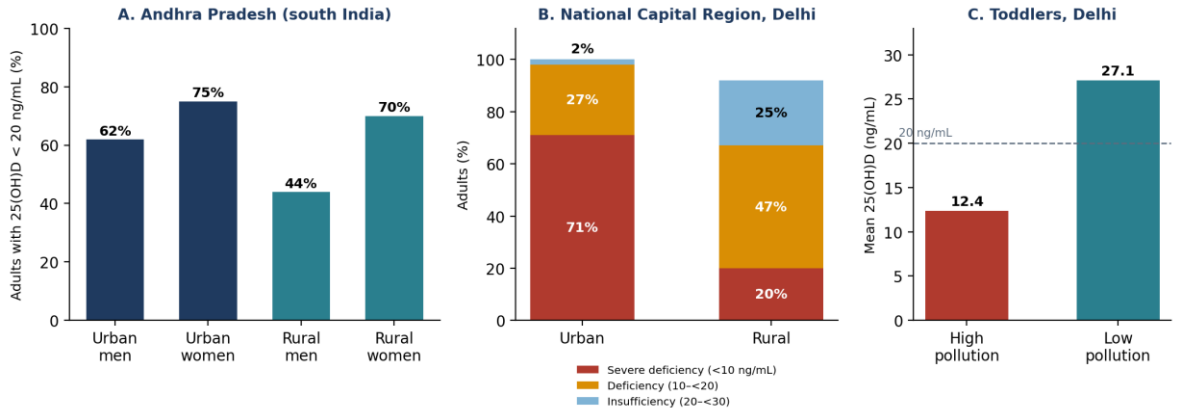


Figure 2. *Selected Indian data on vitamin D status*. (A). *Proportion of healthy adults with 25(OH)D below 20 ng/mL in Andhra Pradesh, by residence and sex* [8]. (B). *Distribution of 25(OH)D categories among adults in the National Capital Region of Delhi* [11]. (C). *Mean 25(OH)D of toddlers living in high- and low-pollution areas of Delhi [13]. Graphs redrawn by the author from the published values*.

Prevalence estimates should be interpreted with care. Most Indian studies are small, regional or hospital-based, they use different assays and cut-offs, and representative national data are lacking [11]. A 2025 report cited in an ICRIER policy brief puts the proportion of deficient Indians at about one in five [12], a figure considerably lower than the older estimates above. Such divergence illustrates that the true national burden is not yet known, even though the evidence of widespread low vitamin D status is consistent across studies.

4. Why Is Deficiency So Common Despite Sunshine?

4.1 Limited effective sun exposure

Plentiful sunshine does not guarantee adequate cutaneous synthesis. Cultural and social practices, clothing, indoor occupations and avoidance of the midday sun reduce effective UVB exposure [1]. Atmospheric pollution adds to the problem. In Delhi, toddlers in a heavily polluted area had much lower 25(OH)D than those in a less polluted area (12.4 *vs* 27.1 ng/mL), with haze scores indicating less UVB at ground level [13].

The role of skin pigmentation is often assumed but is debated. Early work found that heavily pigmented skin synthesised less vitamin D_3 after a standard UV dose [14]. However, Indian and Pakistani immigrants were later reported to have the same capacity as Caucasians to produce vitamin D after UV irradiation [15], and Asians living in Pakistan had 25(OH)D concentrations well within the normal range and significantly higher than Asians living in Britain [16]. Those authors concluded that deficiency in Asians is an environmental rather than genetically determined characteristic [16]. Pigmentation therefore cannot by itself explain the Indian situation.

4.2 Diet and the food system

Few foods are rich in vitamin D, most Indians are vegetarian, and unfortified milk contains very little vitamin D (about 2 IU/100 mL) [1]. The usual Indian diet does not meet the daily vitamin D requirement, and dairy products are rarely fortified [1,2]. Dietary calcium is low and phytate intake is high, so the phytate-to-calcium ratio is unfavourable [8]. Low calcium intake in the presence of vitamin D insufficiency is associated with secondary hyperparathyroidism [1]. Lactose intolerance, high caffeine intake and cooking practices such as prolonged boiling and frying have been proposed as additional dietary contributors [1].

4.3 Host factors and the absence of systematic programmes

Deficiency is more frequent in women, in urban residents and in obese individuals [8,11]. Requirements rise during growth, pregnancy and lactation, and the new-born depends on maternal stores [10]. Finally, India has had no population-wide vitamin D programme: fortification is limited and voluntary, as discussed below [1,12]. Figure 3 summarises these determinants and their consequences.

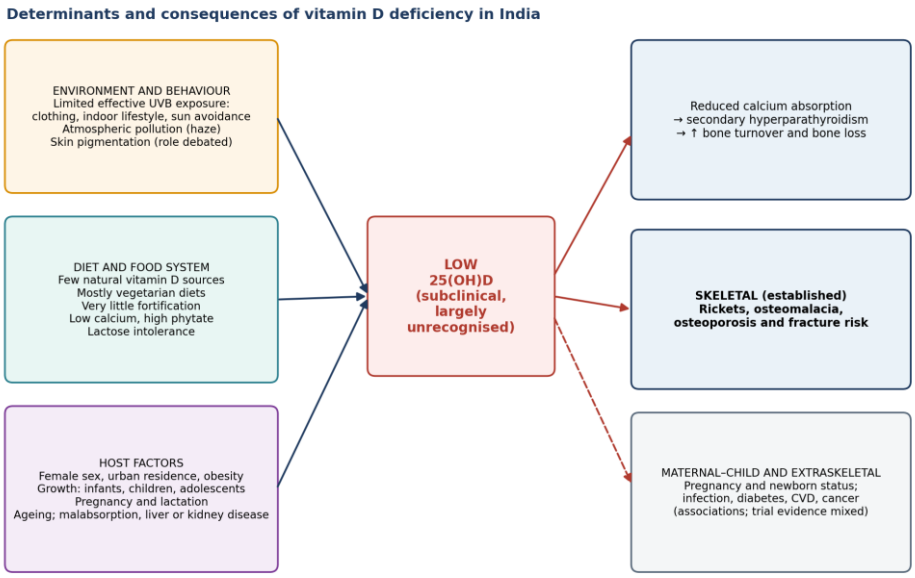


Figure 3. *Determinants and consequences of vitamin D deficiency in India. Solid arrows indicate established relationships; the dashed arrow indicates associations for which trial evidence is mixed. Scheme prepared by the author from refs* [1,4,8,10,11,13–16].

5. Health Consequences and the Strength of the Evidence

5.1 Skeletal and maternal-child outcomes

The skeletal consequences of deficiency are firmly established. Rickets and osteomalacia can be prevented by about 400 IU of vitamin D daily, and 25(OH)D below 50 nmol/L accelerates bone turnover, bone loss and osteoporotic fractures, risks that can be reduced by 800 IU together with adequate calcium in institutionalised or deficient elderly people [4]. Indian studies show the biochemical signature of this process, namely low calcium, raised PTH and chemical osteomalacia [7,8], and clinical signs of deficiency in a tenth of Delhi schoolchildren [9]. Maternal deficiency is directly relevant to the infant, because the foetus and new-born depend on the mother for vitamin D [10].

5.2 Extra-skeletal outcomes

Observational studies link low vitamin D status to infections such as tuberculosis, cardiovascular disease, diabetes and cancer, and it has been proposed that deficiency contributes to the burden of these diseases in India [1]. Randomised evidence is more mixed [4]. An individual-participant meta-analysis of randomised trials found that vitamin D supplementation reduced the risk of acute respiratory infection (adjusted odds ratio 0.88, 95% CI 0.81–0.96), with the greatest benefit in people who were very deficient and in those not receiving bolus doses [17]. In contrast, the large VITAL trial (2000 IU/day in adults in the United States) found no reduction in invasive cancer or major cardiovascular events [18]. The D2d trial (4000 IU/day) did not significantly reduce progression to diabetes in high-risk adults who were not selected for vitamin D insufficiency (hazard ratio 0.88, 95% CI 0.75–1.04) [19]. A meta-analysis of 81 randomised trials concluded that supplementation does not prevent fractures or falls and has no clinically meaningful effect on bone density in adults [20].

These null results should be read with caution in the Indian context. The trials mostly enrolled populations that were not deficient at baseline, whereas the one analysis that stratified by baseline status found the largest benefit in the most deficient participants [17]. In the Delhi survey, 71% of urban adults had 25(OH)D below 10 ng/mL [11]. Whether supplementation prevents extraskeletal disease in such severely deficient populations is a hypothesis that remains to be tested, not a conclusion that can be drawn from the existing trials.

Table 2. Health outcomes associated with vitamin D deficiency and the strength of the evidence.

Outcome	Strength of evidence	Key points	Ref.
Rickets and osteomalacia	Established	Prevented by about 400 IU/day; clinical signs of deficiency in 10.8% of Delhi schoolchildren.	[4,9]
Secondary hyperparathyroidism and bone loss	Established	Raised PTH and low calcium in deficient Indian groups; 25(OH)D <50 nmol/L accelerates bone turnover and loss.	[4,7,8]
Pregnancy and newborn status	Strong association	High prevalence of deficiency in mothers and newborns; fetus depends on maternal vitamin D.	[10]
Acute respiratory infection	Moderate (meta-analysis of trials)	Adjusted OR 0.88 with supplementation; largest benefit in very deficient people.	[17]
Type 2 diabetes	Trial evidence null in high-risk adults not selected for deficiency	4000 IU/day: HR 0.88 (0.75–1.04).	[19]
Cancer and cardiovascular disease	Trial evidence null (VITAL)	2000 IU/day did not reduce invasive cancer or major cardiovascular events.	[18]
Fractures, falls and bone density in adults	Meta-analysis of 81 trials: no clinically meaningful effect	Mostly populations not selected for deficiency.	[20]

6. Public Health Response

6.1 Dietary recommendations and clinical guidance

The ICMR-NIN 2020 recommendations set a recommended dietary allowance of 600 IU (15 µg) of vitamin D per day for adults, alongside 1,000 mg of calcium per day [21]. The 2024 Endocrine Society guideline, co-sponsored by the Endocrine Society of India, suggests against routine 25(OH)D testing in the general population and against supplementing healthy adults younger than 75 years above the current recommended intake, while suggesting empiric supplementation for children, pregnant women, adults aged 75 years and older and adults with high-risk prediabetes [5]. The guideline was written for people without established indications for treatment or testing and drew on trials conducted mostly in Western populations [5], so it does not settle the question for a country where deficiency is so common.

6.2 Food fortification

India's food fortification regulations were operationalised in 2016 and notified in 2018. They cover wheat flour and rice (with iron, vitamin B12 and folic acid), double-fortified salt, and milk and edible oil with vitamins A and D, and they introduced the "+F" logo. When the 2018 regulations were announced, about 21% of the organised milk industry and 47% of the packaged refined edible oil industry were fortifying [22]. A 2025 ICRIER policy brief notes that fortification with vitamin D remains voluntary, is permitted only for edible oil and milk, and allows only plant-based sources of vitamin D. It recommends widening the range of fortified products, permitting vitamin D from plant and animal sources, mandating fortification in selected products, and strengthening monitoring [12]. Fortification is not a complete answer: even in the United States and Canada, vitamin D fortification has been only partly effective [1].

6.3 Supplementation, sunlight and surveillance

Infants, children, pregnant and lactating women and older adults are logical priority groups for targeted supplementation, in line with the groups identified in current guidance [5]. Advice on safe sun exposure and measures to reduce air pollution address the environmental causes [13], although optimal sun-exposure recommendations for Indian populations have not been established. Surveillance is the weakest element: a representative national survey using standardised assays and agreed cut-offs is needed [11,12]. Table 3 and Figure 4 summarise the options.

Table 3. Strategies to address vitamin D deficiency in India: current position and priority actions.

Strategy	Current position	Priority actions	Ref.
Dietary recommendations	ICMR-NIN 2020 RDA of 600 IU/day vitamin D and 1,000 mg/day calcium.	Integrate vitamin D and calcium into national dietary guidance and nutrition programmes.	[21]
Fortification	Voluntary; only edible oil and milk may carry vitamin D; plant-based sources only.	Widen food vehicles, permit animal-derived D_3, mandate selected products, monitor coverage.	[12,22]
Supplementation	Practice varies; trial evidence from non-deficient populations.	Target high-risk groups; avoid routine testing and high-dose use in healthy adults.	[5]
Surveillance and research	Representative national data lacking.	National survey with standardised assays and cut-offs; trials in deficient Indian groups.	[11,12]

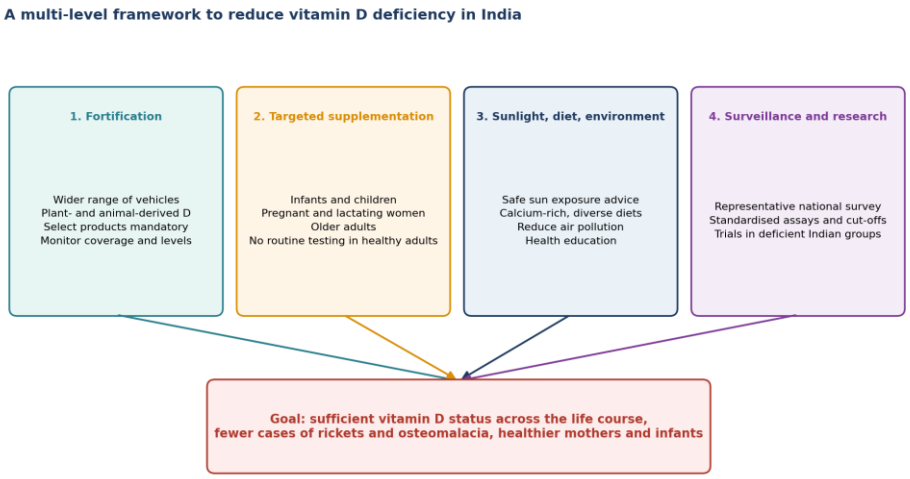


Figure 4. *A multi-level framework to reduce vitamin D deficiency in India, based on refs* [5,12,21,22]. *Scheme prepared by the author.*

7. Challenges and Future Directions

Four gaps stand out. First, India lacks representative national data, and prevalence figures vary widely with population, assay and cut-off [2,11,12]. Second, there is no agreed optimal 25(OH)D target, which complicates both diagnosis and policy [5]. Third, trials are needed in severely deficient Indian populations, because the existing large trials were conducted in groups that were mostly replete [17–19]. Fourth, public attention must be balanced: the benefit-risk ratio of the recent rise in vitamin D testing and use is not clear [5], so the response to a real deficiency problem should not become indiscriminate testing and high-dose supplementation.

8. Conclusions

Low vitamin D status is common in India across age groups, regions and income levels, and it is a plausible contributor to the burden of rickets, osteomalacia and poor maternal and infant health. Its causes lie less in a lack of sunshine than in how people live and eat, and the absence of fortification and targeted programmes. The evidence for benefits outside the skeleton is mixed, and the existing trials cannot answer the question for severely deficient populations. A coordinated response that combines a national survey with standardised measurement, broader fortification and targeted supplementation for high-risk groups is warranted, together with trials in Indian populations.

Declaration, Conflict of interest and funding

The content provided herein is developed using AI tools, and is meant to help Researchers who are working in the fields of Medical & Health Sciences.

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Data availability: No new datasets were generated or analyzed.

Author Note

This manuscript is a literature review and is not a treatment recommendation for any individual patient. Vitamin D supplementation should be taken only for a clinically justified indication and with appropriate medical monitoring under the supervision of an expert physician.

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