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"RECONSTRUCTING THE VITAMIN D PARADIGM: FROM DIETARY GAPS TO FORTIFIED FUTURES"

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ABSTRACT

Vitamin D, a fat-soluble secosteroid hormone, plays a crucial role in calcium-phosphorus homeostasis, bone mineralization, immune modulation, and cellular growth regulation. The primary sources are ultraviolet B (UVB)-induced cutaneous production and a few dietary sources, including fortified meals, fatty fish, liver, and egg yolks makes it both accessible and elusive, depending on lifestyle, geography, and cultural factors. The active form of 1,25-dihydroxyvitamin D [1,25(OH)$_2$D] is produced by the hepatic and renal hydroxylation of vitamin D. It binds to the vitamin D receptor (VDR) to regulate gene expression and preserve systemic mineral balance.

Vitamin D status can still be accurately determined by measuring serum 25(OH)D concentration. When a deficit is indicated by levels below 20 ng/ml (50 nmol/L). According to the Recommended Dietary Allowance (RDA), which normally ranges between 600 and 800 IU per day. Despite its well-established importance, vitamin D deficiency has spread like an epidemic, affecting an estimated one billion people worldwide. Malabsorption, obesity, ageing, greater skin melanin content, poor solar exposure, and inadequate food intake are all contributing causes.

Deficiency consequences are extensive, encompassing rickets in children, osteomalacia and osteoporosis in adults, increased fracture risk, autoimmune disorders, cardiovascular diseases, infections, insulin resistance, and neurocognitive decline. In response, food fortification has emerged as a public health strategy. In India, common fortified foods include milk, edible oils, cereals, and condiments. However, excessive intake, especially through high-dose supplementation, may lead to toxicity, manifesting as hypercalcemia, nephrocalcinosis, and vascular calcification.

In conclusion, minimising widespread deficiency and related health consequences requires optimising vitamin D status through dietary sources, safe sun exposure, and suitable fortification. Evidence-based supplementation and fortification techniques ought to be the focus of future regulations.

KEY WORDS: -

Secosteroid, 1,25-dihydroxyvitamin D, Dietary sources, Fortified foods, Toxicity

INTRODUCTION

Vitamin D is a fat-soluble vitamin that is essential for numerous physiological functions, particularly in bone health, immune modulation, and cellular growth. It is different from other

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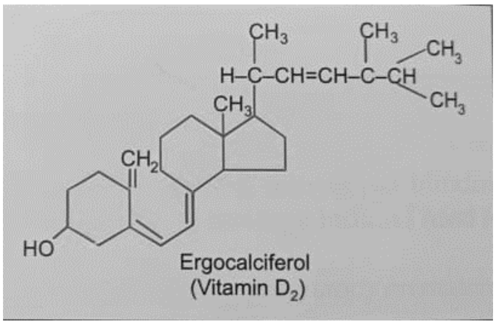
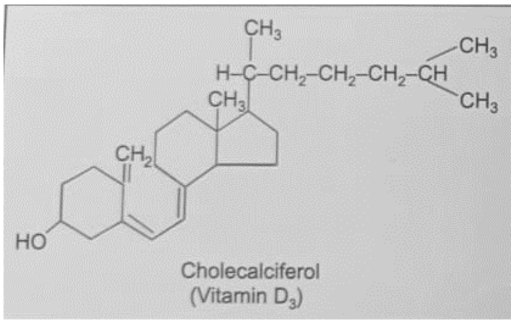
vitamins in that it is a nutrient and a precursor to hormones because the human body can produce it when the skin is exposed to ultraviolet B (UVB) radiation from sunlight (Holick, 2007) [1].

The two primary forms of vitamin D are cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2). Vitamin D3 is derived from animal-based foods and is also the form that the skin synthesises, whereas vitamin D2 is obtained from plant sources and fortified foods. It was in 1922 that the precursors of vitamin D, 7-dehydrocholestrol in animals, and ergosterol in plants were found. By 1936, the molecular structure of vitamin D has been established after it was isolated in crystalline form in 1932 (Sri Lakshmi, B. 2019) [2].

Chemical Structure of Vitamin D2 and D3 given below (Sri Lakshmi, B. 2019) [2].



Chemical Structure of Vit D2



Chemical Structure of Vit D3

When it comes to increasing and sustaining serum 25-hydroxyvitamin D concentrations over a longer period of time, vitamin D3 is at least three times more effective than vitamin D2 (Sri Lakshmi, B. 2019) [2].

According to Deluca (2004), both forms are physiologically inactive and must undergo hydroxylation in the liver and kidneys in order to transform into the active form, calcitriol (1,25-dihydroxyvitamin D) [3].

Maintaining calcium and phosphorus homeostasis, which is essential for bone formation and skeletal health, is one of vitamin D's main functions. It promotes the remodeling and mineralisation of bone tissue, improves intestinal absorption of calcium and phosphorus, and makes it easier for the kidneys to reabsorb these nutrients (Bouillon et al., 2008) [4]. Vitamin D deficiency can cause osteomalacia in adults, which causes bone pain and muscle weakness, and rickets in children, which is characterised by abnormalities of the bones (Holick, 2007) [1]. Furthermore, osteoporosis and fractures are linked to chronic vitamin D deficiency, especially in older adults (Dawson-Hughes et al., 2005) [5].

Vitamin D is essential for immune system modulation in addition to its skeletal roles. By controlling the activity of immune cells like T and B lymphocytes, dendritic cells, and macrophages, it affects both the innate and adaptive immune responses (Aranow, 2011) [6]. According to new research, having enough vitamin D may improve immunity to infections and lower the risk of autoimmune conditions like rheumatoid arthritis and multiple sclerosis (Sassi

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et al., 2018). Furthermore, studies have linked vitamin D to the control of cell differentiation and proliferation, suggesting that it may lower the risk of developing certain cancers, such as breast and colorectal cancer (Holick, 2011) [7].

The main sources of vitamin D in the diet are fatty fish (such as salmon, mackerel, and sardines), fish liver oils, egg yolks, and fortified foods (Ross et al., 2011) [8]. Sunlight exposure, however, continues to be the primary source for the majority of people. Geographic latitude, season, skin pigmentation, and sunscreen use are some of the variables that affect the skin's production of vitamin D (Holick, 2004) [9]. People with darker skin or those who live in higher latitudes may be less able to synthesise vitamin D, which increases their risk of deficiency.

According to estimates, more than one billion people worldwide lack adequate amounts of vitamin D, making it a global public health concern (Holick, 2007) [1]. Numerous medical conditions, such as cardiovascular disease, type 2 diabetes, and neurodegenerative disorders, are linked to deficiencies (Pilz et al., 2011) [10]. Age, sex, and life stage all affect the Recommended Dietary Allowance (RDA) for vitamin D. According to current recommendations, most adults should consume 600–800 IU daily, while older adults and those with known deficiencies should take higher doses (Ross et al., 2011) [8].

SOURCE OF VITAMIN D

Vitamin D is naturally present in only a limited number of foods, making it challenging to meet daily requirements through diet alone. It exists in two biologically relevant forms: **Vitamin D_2 (ergocalciferol)**, obtained from plant and fungal sources, and **Vitamin D_3 (cholecalciferol)**, synthesized endogenously in human skin upon exposure to ultraviolet B (UVB) radiation and derived from animal-based foods (Holick, 2007) [1]. Fatty fish including salmon, mackerel, sardines, and tuna, as well as cod liver oil, one of the most concentrated dietary sources, are rich natural sources of vitamin D_3 (NIH, 2023) [11]. Dairy fats such butter and ghee, cow liver, and egg yolks are additional moderate sources (ICMR-NIN, 2020) [12]. Many nations fortify basic items including milk, plant-based beverages (such soy or almond milk), breakfast cereals, yoghurt, and margarine with either D_2 or D_3 to increase public intake because natural vitamin D-rich foods are scarce (Calvo et al., 2005) [13]. UV-exposed mushrooms are a special plant source of vitamin D_2 for vegetarians and vegans, and depending on exposure duration and intensity, they can supply substantial amounts (Lu et al., 2007) [14]. Despite these alternatives, widespread vitamin D insufficiency and inadequacy are still caused by low dietary consumption and limited sun exposure in some areas, highlighting the significance of fortified foods and supplements for preserving normal vitamin D status.

Food Source of Vitamin D

Food Item	**Serving Size**	**Vitamin D Content**
Cod liver oil	1 tbsp (15 mL)	1,360 IU (34 mcg)
Salmon (cooked, wild)	100 g	600–1,000 IU (15–25 mcg)
Mackerel (cooked)	100 g	~360 IU (9 mcg)
Sardines (canned in oil)	2 fish (75 g)	~250 IU (6.3 mcg)
Tuna (canned in water)	100 g	~236 IU (5.9 mcg)

Egg yolk	1 large	~40–50 IU (1–1.25 mcg)
Beef liver (cooked)	100 g	~50 IU (1.25 mcg)
Butter/ghee	10 g	~15–20 IU (0.4–0.5 mcg)
UV-exposed mushrooms	100 g	400–1,000 IU

MECHANISM OF ABSORPTION OF VITAMIN D

Vitamin D, a fat-soluble (lipophilic), is primarily obtained from dietary sources (D_2 from plants and D_3 from animal products) and skin synthesis upon exposure to ultraviolet B (UVB) radiation.

The ileum and jejunum are the main digestive organs where dietary vitamin D is absorbed. Because it is a lipophilic molecule, bile salts and dietary fats are essential for its absorption because they emulsify it and help mixed micelles form. Via passive diffusion or aided transport via certain lipid transporters such as SR-BI (scavenger receptor class B type I), NPC1L1 (Niemann–Pick C1-like 1), and CD36, these micelles enable vitamin D to permeate across the apical membrane of enterocytes (Reboul et al., 2006) [15]. Triglycerides, cholesterol esters, and vitamin D are all integrated into chylomicrons once they are inside the enterocyte. After entering the lymphatic system, these chylomicrons travel through the thoracic duct to the systemic circulation.

Vitamin D is mainly attached to vitamin D-binding protein (DBP) in the bloodstream, which carries it to the liver, where the initial metabolic activation phase takes place. The primary circulating and storage form of vitamin D is 25-hydroxyvitamin D [25(OH)D], which is created in the liver when 25-hydroxylase enzymes (mainly CYP2R1) hydroxylate vitamin D. The enzyme 1α-hydroxylase (CYP27B1) must further hydroxylate this physiologically inactive metabolite in the kidney's proximal tubules to create 1,25-dihydroxyvitamin D [$1,25(OH)_2D$], often referred to as calcitriol, the hormonally active version. The ligand-receptor complex that forms when calcitriol binds to vitamin D receptors (VDR) in the intestinal, bone, kidney, and immune cells moves to the nucleus, where it controls the transcription of genes related to immune modulation, cell differentiation, and calcium and phosphate homeostasis. (Haussler et al., 2011; Jones, 2008) [16,17].

SERUM CONCENTRATION LEVEL OF VITAMIN D IN BODY

The serum amounts of 25(OH)D linked to deficiencies, enough for bone health, and optimal general health are strongly contested, and no scientific consensus procedure has established cut points. A committee of the Institute of Medicine determined that individuals are at risk of vitamin D deficiency at serum 25(OH)D concentrations <25 nmol/L (<10 ng/mL) after reviewing data on vitamin D needs. Some may be at risk for insufficiency at concentrations between 25 and 49 nmol/L (10 and 19 ng/mL). Nearly everyone is adequate at levels 50-74 nmol/L (20-29 ng/mL); according to the committee, the serum 25(OH)D level that meets the requirements of 97.5% of the population is 75-125 nmol/L. Serum levels more than 125 nmol/L (>50 ng/mL) are linked to possible negative consequences (Institute of Medicine. 2011) [18].

Serum 25(OH)D Level	nmol/L	ng/mL	Health Interpretation	Health Implications

Severely Deficient	< 25	< 10	Severe deficiency	Rickets in children, osteomalacia in adults, muscle weakness, immune dysfunction
Deficient	25–49	10–19	Deficiency	Increased risk of osteoporosis, bone pain, fractures, falls, and possible autoimmune dysfunction
Insufficient	50–74	20–29	Insufficiency	Suboptimal bone and muscle health; risk of chronic diseases remains elevated
Sufficient (Optimal)	75–125	30–50	Optimal/sufficient	Adequate for bone health, calcium absorption, immune function, and chronic disease prevention
Potentially Toxic	125–150	50–60	Risk of toxicity	Associated with hypercalcemia, nephrocalcinosis, renal failure, and vascular calcification

REFERENCE INTAKE

- Recommended Dietary Allowance (RDA): average daily level of intake sufficient to meet the nutrient requirements of nearly all (97%–98%) healthy people.
- Adequate Intake (AI): established when evidence is insufficient to develop an RDA and is set at a level assumed to ensure nutritional adequacy.
- Tolerable Upper Intake Level (UL): maximum daily intake unlikely to cause adverse health effects

Recommended Dietary Allowances (RDAs) for Vitamin D [19,20]

Age Group	RDA (IU/day)	RDA (mcg/day)
Infants (0–6 months)	400 IU	10 mcg
Infants (7–12 months)	400 IU	10 mcg
Children (1–3 years)	600 IU	15 mcg
Children (4–8 years)	600 IU	15 mcg
Children & Teens (9–18 years)	600 IU	15 mcg
Adults (19–50 years)	600 IU	15 mcg
Adults (51–70 years)	600 IU	15 mcg
Older Adults (71+ years)	800 IU	20 mcg
Pregnant Women (All ages)	600 IU	15 mcg
Lactating Women (All ages)	600 IU	15 mcg

UNITS 9Sri Lakshmi, B. 2019) [2]

1µg of vitamin D = 40 IU of vitamin D

1 IU of Vitamin D = 25 Nanogram (25Ng) of Vitamin D

1µg of 1, 25 dihydroxy vitamin D = 1000 IU of Vitamin D

PREVALENCE OF VITAMIN D DEFICIENCY

The prevalence of vitamin D deficiency is influenced by a variety of factors, including geographical location, lifestyle choices, age, skin pigmentation, dietary habits, and health conditions. According to research, vitamin D deficiency is extremely prevalent throughout the world, with notable regional variations. Nearly 1 billion people worldwide are thought to be deficient in vitamin D, according to a systematic review and meta-analysis by Palacios and Gonzalez (2014) [21]. The prevalence may be higher than 70% in some populations, especially those with darker skin tones, those who live in higher latitudes, and those who receive less sun exposure. These individuals also have a decreased capacity to synthesise vitamin D from sunlight.

According to reports, the prevalence of vitamin D deficiency in the general population in nations like the US is between 20 and 30 percentage; however, this rate rises for certain demographics, including older adults, obese people, and people with certain chronic illnesses (Holick, 2007). According to research studies, up to 50% of people in Europe suffer from low vitamin D levels; in northern nations with less sunlight, the prevalence is even higher (Wang et al., 2018) [22].

The prevalence is frequently higher in Asia, estimates indicate that up to 70% of people in nations like China, India, and Malaysia may be vitamin D deficient as a result of cultural customs, high air pollution levels, and inadequate dietary intake (Zaidi, 2015) [23]. Vitamin D deficiency is also very common in Africa, especially in urban areas where people spend less time outside and may avoid sun exposure because of cultural norms (Kesson et al., 2021) [24].

India is not an exception to the growing global public health concern over vitamin D deficiency. India is seeing shockingly high rates of vitamin D deficiency in a number of populations, despite having an abundance of sunlight. Limited sun exposure, dietary habits, urbanisation, and cultural customs are some of the causes of this paradoxical scenario.

According to studies, vitamin D deficiency is extremely prevalent in India; estimates indicate that more than 70% of the population may not have adequate amounts of this vital nutrient (Sharma et al., 2018) [25]. According to a study by Sahota et al. (2017) [26], 60% of Indians living in rural areas and nearly 80% of those living in cities had inadequate vitamin D levels. Because their bodies are less able to produce vitamin D through exposure to sunlight, women, children, the elderly, and people with darker skin tones are more likely to suffer from this deficiency.

The prevalence of vitamin D deficiency differs significantly by region. Deficiency rates are higher in northern and central India, where winters are cold and sunlight is scarce for several months. On the other hand, because of lifestyle choices like working indoors and wearing sunscreen frequently, the prevalence is still high in southern states, where sunlight exposure is more constant (Sakthivel et al., 2020) [27].
The high incidence of vitamin D deficiency in India is caused by a number of lifestyle factors. Most significantly, the problem has been made worse by the tendency towards indoor living, overuse of sunscreen, and insufficient intake of foods high in vitamin D, such as eggs, fish, and dairy products with added vitamin D. Furthermore, cultural customs that promote modest attire reduce exposure to direct sunlight, which exacerbates the deficiency issue (Chandran et al.,2021) [28].

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In children, low levels of vitamin D have been linked to poor bone health and an increased risk of rickets, while in adults, deficiency has been associated with osteoporosis, cardiovascular diseases, and autoimmune disorders (Vaswani et al., 2019) [29]. As the wider negative health effects of vitamin D become more widely recognised, there is a growing demand for public health interventions, including supplementation and fortification programs, especially for vulnerable populations.

CAUSES OF VITAMIN D DEFICIENCY

Vitamin D deficiency is a significant public health issue in India, even though the country receives abundant sunlight throughout the year.

SUN EXPOSURE

Vitamin D deficiency is primarily caused by inadequate exposure to sunlight, specifically ultraviolet B (UVB) radiation, which is essential for the synthesis of vitamin D in human skin. When bare skin is exposed to ultraviolet (UV) B) light with a wavelength of 290–320 nanometres, cutaneous 7-dehydrocholesterol is transformed into previtamin D3, which is then transformed into vitamin D3. Sunscreen, cloud cover, smog, skin melanin concentration, season, time of day, and length of day are some of the elements that influence UV radiation exposure and vitamin D production. The average blood 25(OH)D levels in a population are not always predicted by geographic latitude, which may come as a surprise. Due to sedentary lifestyles, work, and education, many people in urban areas spend the majority of their time indoors. Opportunities for natural sunlight exposure, which is crucial for the skin's production of vitamin D, are further diminished by the growing preference for indoor recreation and air-conditioned spaces (Agarwal et al., 2021) [30]. In addition to blocking the skin's capacity to absorb UVB rays, high-SPF sunscreens can also hinder the synthesis of vitamin D (Norval & Wulf, 2009).

SKIN TONE

Skin tone has a major effect on the synthesis of vitamin D. The skin of many Indians is darker, meaning it contains more melanin. Melanin decreases the skin's capacity to effectively produce vitamin D, even though it aids in protecting against ultraviolet (UV) damage. The amount of UVB rays that reach the skin is further reduced by the widespread use of sunscreens brought on by increased awareness of tanning and skin damage (Ramasamy et al., 2020) [32].

CLOTHING

Clothing choices and cultural customs have a big impact on vitamin D levels as well. The majority of the body is usually covered by traditional clothing, particularly for women, such as sarees, salwar kameez, burqas, and long sleeves. By limiting skin exposure to sunlight, this clothing lowers the amount of vitamin D that is naturally synthesised. (Joshi and Harinarayan, 2019) [33].

DIETARY HABITS

Dietary habits also contribute to the problem. A lot of Indians eat plant-based or vegetarian diets, which inherently don't contain enough vitamin D. Vegetarian diets either lack or consume insufficient amounts of foods high in vitamin D, such as oily fish, egg yolks, and fortified dairy products. The lack of widespread fortification of common foods in India exacerbates the deficiency (Kalra et al., 2021) [31].

POLLUTION

Air pollution is another important but frequently neglected factor. Extremely high levels of air pollution prevent ultraviolet B (UVB) rays from reaching the ground in places like Delhi, Mumbai, and Kolkata. Because UVB rays are necessary for the synthesis of vitamin D, air pollution significantly impairs the skin's capacity to generate sufficient amounts of the vitamin (Mishra et al., 2020) [34].

OTHER FACTORS

Biological factor that contributes to vitamin D deficiency is obesity. Being fat-soluble, vitamin D tends to become trapped in fat tissues in obese people, reducing its bioavailability for body processes. Additionally, the body's capacity to absorb vitamin D from food and supplements is hampered by a number of gastrointestinal disorders, including Crohn's disease, coeliac disease, and irritable bowel syndrome (IBS) (Sachan et al., 2019) [35]. Lastly, the Indian populace generally lacks knowledge regarding the significance of vitamin D. Until symptoms worsen, people frequently are unaware that they may be lacking. Furthermore, routine screening for vitamin D deficiency is not widely done, particularly among economically disadvantaged groups and in rural areas. As a result, the condition often remains undiagnosed and untreated for long periods (Puri et al., 2018) [36].

CONSEQUENCES OF VITAMIN D DEFICIENCY

Vitamin D deficiency can have serious health consequences that affect multiple systems in the body.

OSTEOMALACIA AND RICKETS

A lack of vitamin D significantly reduces the intestinal absorption of calcium and phosphorus, which causes osteomalacia in adults and rickets in children. Osteomalacia results in bone softness, muscle weakness, and a higher risk of fractures because of poor bone mineralization, whereas rickets presents with skeletal abnormalities, delayed growth, and pain (Holick, 2007) [1].

FRACTURES AND OSTEOPOROSIS

Chronic vitamin D insufficiency increases bone fragility and decreases bone mineral density, which leads to osteoporosis, particularly in the elderly. Because of decreased calcium deposition and bone remodeling, this disease increases the risk of fractures, especially in the hip and spine (Bischoff-Ferrari et al., 2005) [37].

MUSCLE WEAKNESS AND FALLS

especially in older adults, proximal muscle weakness is associated with vitamin D deficiency, which can result in poor postural balance and an increased risk of falls. In deficiency circumstances, muscle strength and coordination are affected by vitamin D receptors in muscle tissue (Pfeifer et al., 2000) [38].

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REDUCED IMMUNE RESPONSE

Immune modulation is negatively impacted by vitamin D insufficiency, which impairs both innate and adaptive immunity. It makes people more vulnerable to infections like influenza, TB, and respiratory tract diseases. This is because fewer antimicrobial peptides, such as cathelicidin, are being produced (Aranow, 2011) [39].

CARDIOVASCULAR DISEASE

Heart failure, atherosclerosis, and hypertension are all linked to low serum 25(OH)D levels. A lack of vitamin D raises the risk of cardiovascular events by promoting vascular inflammation and activating the renin-angiotensin-aldosterone pathway (Wang et al., 2008) [40].

INSULIN RESISTANCE AND DIABETES

Insulin receptor expression and pancreatic β-cell activity are both impacted by vitamin D. By causing insulin resistance and decreased glucose tolerance, deficiencies raise the risk of type 2 diabetes and metabolic syndrome (Chiu et al., 2004) [41].

AUTOIMMUNE DISEASES

Vitamin D lowers the synthesis of inflammatory cytokines and regulates T-cell responses. A higher incidence of autoimmune diseases such multiple sclerosis, rheumatoid arthritis, and type 1 diabetes is associated with its deficiency (Holick, 2007) [1].

ADVERSE PREGNANCY OUTCOMES

Vitamin D deficiency during pregnancy increases the risk of preterm birth, low birth weight babies, gestational diabetes, and preeclampsia. Both maternal health and the formation of fetal bone depend on adequate amounts (Bodnar et al., 2007) [42].

VITAMIN D FORTIFIED FOODS AVAILABLE IN INDIA

In India, vitamin D deficiency is very common because of things like low food intake, little sun exposure, and high skin melanin content, which lowers cutaneous production (Harinarayan & Joshi, 2009) [43]. Food fortification has become a public health technique that has been approved by the Food Safety and Standards Authority of India (FSSAI) to address widespread deficiency. As component of its "+F" branding project, FSSAI suggests adding vitamin D to regularly consumed staple foods like rice, milk, edible oil, and wheat flour (atta).

Fortified Atta (Wheat Flour)		
Brand	**Product**	**Vitamin D Content**
Aashirvaad	Fortified Whole Wheat Atta	1.25 mcg/100 g (~50 IU)
Patanjali	Fortified Atta	Enriched with Vitamin D & Iron
Fortified Milk & Dairy Products		
Brand	**Product**	**Vitamin D Content**
Amul	Amul Taaza Fortified Milk	45 IU/100 mL
Mother Dairy	Fortified Toned Milk	42 IU/100 mL
Nestlé	Nestlé a+ Milk	40–50 IU/100 mL
Britannia	Milk Booster for Children	Fortified with Vitamin D & Calcium
Danone (Protinex)	Protinex Powder	100 IU/serving (approx.)
Fortified Edible Oils		
Brand	**Product**	**Vitamin D Content**
Fortune	Vitamin-Fortified Sunflower Oil	20 IU/10 g

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Dhara	Fortified Vegetable Oil	Vitamin A & D fortified
Saffola	Fortified Edible Oil (Active)	Contains added Vitamin D
Fortified Ready-to-Eat Foods & Beverages		
Brand	**Product**	**Vitamin D Content**
Kellogg's	Cornflakes, Muesli	80–100 IU per serving
Bagrry's	Crunchy Muesli, Bran Flakes	Fortified with Vitamin D
Horlicks	Classic, Women's, Junior	200–400 IU per serving
Bournvita	Bournvita Health Drink	Fortified with D & Calcium
Pediasure	Child Nutrition Drink	100–150 IU per serving
Ensure	Adult Nutritional Supplement	200–400 IU per serving
Boost/Complan	Health & Growth Beverages	Fortified with Vitamin D

(FSSAI 2023, ICMR-NIN 2020, WHO/FAO 2006, NIH Office of Dietary Supplements 2023 [44,45,46,47])

HEALTH RISKS FROM EXCESSIVE VITAMIN D INTAKE

vitamin D is essential for calcium homeostasis and bone health, excessive consumption of it, especially from high-dose supplements or prolonged over-fortification, can make it hazardous. The main pathophysiological consequence of vitamin D toxicity, sometimes referred to as hypervitaminosis D, is hypercalcemia, which is defined as blood calcium levels greater than 10.5 mg/dL (Holick, 2007; Vieth, 1999) [1,48]. Bypassing the typical feedback inhibition, high 25-hydroxyvitamin D [25(OH)D] increases intestinal calcium absorption beyond physiological control, which is why this happens. Anorexia, nausea, vomiting, constipation, polyuria, and polydipsia are among the early signs of hypercalcemia. These symptoms can lead to mental disorders like disorientation, sadness, and in extreme situations, stupor or coma (Jones, 2008).

Long-term hypercalcemia can cause soft tissues, such as the heart, lungs, kidneys, and blood vessels, to become calcified. This can lead to kidney stones, nephrocalcinosis, or even chronic kidney impairment. Ironically, too much vitamin D can also harm bone health by upsetting the calcium, phosphorus, and vitamin K2 balance, which causes bone demineralisation and fragility. Other severe side effects linked to long-term toxicity include arterial calcification and cardiac arrhythmias. (Holick, 2007) [1].

The **tolerable upper intake level (UL)** for vitamin D is set at **4000 IU/day (100 µg/day)** for adults and children over 9 years, while toxicity generally occurs at sustained intakes **above 10,000 IU/day** (IOM, 2011) [49]. Notably, exposure to sunshine cannot cause vitamin D toxicity since UVB-induced cutaneous synthesis is strictly controlled and self-limiting (Holick, 2007) [1]. However, supplement toxicity can happen subtly, particularly when people take large dosages of supplements for self-medication or when compounded formulations are not dosed correctly. Clinical examination is necessary for high serum 25(OH)D concentrations over 150 ng/mL (375 nmol/L), which are deemed potentially hazardous (Ross et al., 2011; Bouillon et al., 2019). Furthermore, despite early increases in serum calcium, too much vitamin D may interfere with vitamin K-dependent calcium control, paradoxically encouraging bone demineralisation and raising the risk of fractures over time (Schurgers et al., 2007).

Tolerable Upper Intake Level (UL) of Vitamin D

According to the **Institute of Medicine (IOM)** [49] and **EFSA**, the **upper limit** of vitamin D intake is:

Age Group	**Tolerable Upper Intake (per day)**
Infants (0–12 months)	1000–1500 IU
Children (1–8 years)	2500–3000 IU
Adults (≥9 years)	4000 IU
Pregnant/Lactating women	4000 IU

CONCLUSION

Vitamin D plays a crucial role in maintaining bone health, supporting the immune system, and promoting overall physical and mental well-being. It helps the body's absorption of calcium and phosphorus, which are necessary for the development and maintaining of healthy bones and teeth. Vitamin D affects immune responses, muscle function, and even mood regulation in addition to its benefits for the skeleton. Despite its significance, vitamin D deficiency continues to be a global public health issue that impacts individuals of all ages. Limited exposure to sunlight, the main natural source of vitamin D, is one of the most frequent causes of this deficiency. Many people wear clothes that limit their exposure to the sun, or spend the majority of their time indoors due to work or lifestyle choices, which impairs the body's ability to produce adequate vitamin D through the skin. Another significant factor is inadequate dietary intake, particularly for vegetarians and vegans whose natural food sources of vitamin D are primarily animal-based. A higher risk of deficiency may also result from problems absorbing or converting vitamin D into its active form in people with specific medical conditions, such as obesity, liver or kidney disease, and gastrointestinal disorders. Low vitamin D levels can have major and long-lasting effects. It raises the risk of fractures by causing bone-related disorders like rickets in children and osteomalacia or osteoporosis in adults. Muscle weakness, falls in the elderly, weakened immunity, diabetes, cardiovascular disease, and other metabolic disorders are also associated with deficiencies.

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