



Association Between Vitamin D Deficiency And Sedentary Lifestyle Among Females

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Abstract: Vitamin D deficiency in women is a pervasive health concern prevalent among women with far-reaching implications for overall well-being. The present article provides a concise overview of the prevalence of vitamin D deficiency among women and its association with lifestyle factors. Factors contributing to its prevalence include limited sun exposure, dietary choices, and hormonal changes associated with pregnancy and menopause. According to available data from various research papers, the Present article explores the extent of deficiency in various regions and age groups, underlining the necessity for targeted interventions. Lifestyle plays a pivotal role in vitamin D status for women. Inadequate sun exposure, stemming from indoor occupations and cultural practices, remains a primary contributor. Dietary preferences, often lacking in calcium-rich foods, further exacerbate the problem. Lifestyle-related factors like obesity can compromise vitamin D bioavailability. Vitamin D deficiency in women is associated with an increased risk of osteoporosis, autoimmune diseases, and mood disorders. Promoting lifestyle modifications, including sun exposure, dietary improvements, and supplementation, is essential in addressing this concern. Awareness campaigns and tailored strategies are pivotal in the prevention and management of vitamin D deficiency.

Index Terms - Vitamin D, Diet, Lifestyle, Sun exposure, Prevalence

I. INTRODUCTION

Vitamin D deficiency has emerged as a pervasive nutritional concern in India, exhibiting a staggering prevalence that remains widely undiagnosed and untreated. Regardless of factors such as gender, age, sex, race, and geography, individuals across the country are grappling with the adverse effects of this deficiency. Beyond its established role in skeletal health, recent research has illuminated the intricate association between vitamin D deficiency and a spectrum of disorders. In adults, chronic deficiency contributes to conditions like osteomalacia, osteoporosis, and muscle weakness, amplifying the urgency to address this silent epidemic.^[i]

Vitamin D is essential for protecting the body from infections, inflammation, and some cancers.^[i] As a fat-soluble vitamin, it regulates calcium balance and bone health. Deficiency causes rickets in children and osteomalacia in adults. Vitamin D fortification of milk in the 1930s eliminated rickets, yet subclinical deficiency remains common worldwide, affecting up to 1 billion people.^[ii]

The active form, 1,25-dihydroxy vitamin D, influences over 200 genes involved in cell growth, specialization, death, and blood vessel formation, making it vital for cellular balance and overall health.^[iii]

Moreover, vitamin D emerges as a sentinel in the maintenance of extracellular calcium levels, further underlining its indispensability in cellular dynamics. This dual role, as both a genetic modulator and a guardian of calcium homeostasis, underscores the significance of 1,25-dihydroxy vitamin D in the intricate dance of cellular activities. The comprehensive understanding of these molecular intricacies provides a compelling avenue for further research, offering insights into potential therapeutic applications and advancing our comprehension of cellular biology.ⁱⁱ Subclinical vitamin D deficiency is linked to osteoporosis, heightened fall and fracture risks. Recent studies conflict on its associations with cancer, cardiovascular disease, diabetes, autoimmune conditions, and depression. As a research scholar, navigating these complexities is crucial for understanding the multifaceted impact of vitamin D on health.ⁱⁱⁱ

India's unique cultural and social landscape further complicates the scenario, influencing lifestyle choices with pervasive taboos. A predominant vegetarian diet, coupled with socioeconomic disparities, underscores the challenges faced by a significant portion of the population. Notably, women of childbearing age are particularly susceptible to the detrimental effects of vitamin D deficiency and low serum calcium levels. Recognizing the multifaceted implications, our research aims to meticulously estimate the prevalence of serum vitamin D and calcium deficiencies and insufficiencies. By identifying associated risk factors, we strive to shed light on the intricate interplay between cultural, social, and nutritional determinants, offering a comprehensive understanding of this pressing public health issue.^{iv}

Etiology of Vitamin D:-

Dermal synthesis and dietary intake (fatty fish livers, fortified food) are the major sources of ergocalciferol (D2) and cholecalciferol (D3), both of which are converted to 25-hydroxy-vitamin D2 (25-OH-D2) and 25-hydroxy-vitamin D3 (25-OH-D3) respectively in the liver by the enzyme hepatic enzyme 25-hydroxylase. 25-OH-D2 and 25-OH-D3 are then converted to the most active form of vitamin D (1,25 dihydroxyvitamin D) by the enzyme 1-alpha-hydroxylase in the kidneys. This active 1,25 dihydroxyvitamin D increases intestinal absorption of calcium and bone resorption and decreases renal excretion of calcium and phosphate. Vitamin D deficiency can result from several causes.

1. Decreased dietary intake and/or absorption

Certain malabsorption syndromes such as celiac disease, short bowel syndrome, gastric bypass, inflammatory bowel disease, chronic pancreatic insufficiency, and cystic fibrosis may lead to vitamin D deficiency. Lower vitamin D intake orally is more prevalent in the elderly population.^v

2. Decreased sun exposure

Twenty minutes of sunshine daily with over 40% of skin exposed is required to prevent vitamin D deficiency.^{vi} Cutaneous synthesis of vitamin D declines with aging. Dark-skinned people have less cutaneous vitamin D synthesis. Decreased exposure to the sun, as seen in individuals who are institutionalized or have prolonged hospitalizations, can also lead to vitamin D deficiency.^{vii} Effective sun exposure is decreased in individuals who use sunscreens consistently.

3. Decreased endogenous synthesis

Individuals with chronic liver disease such as cirrhosis can have defective 25-hydroxylation, leading to deficiency of active vitamin D. Defects in 1-alpha 25-hydroxylation can be seen in hyperparathyroidism, renal failure, and 1-alpha hydroxylase deficiency.

4. Increased hepatic catabolism

Medications such as phenobarbital, carbamazepine, dexamethasone, nifedipine, spironolactone, clotrimazole, and rifampin induce hepatic p450 enzymes, which activate the degradation of vitamin D.

5. End organ resistance

End-organ resistance to vitamin D can be seen in hereditary vitamin D-resistant rickets.^{viii}

Epidemiology of Vitamin D:-

Vitamin D deficiency is a widespread global health issue, affecting about 1 billion people, with 50% of the population experiencing insufficiency. High-risk groups include the elderly, obese individuals, nursing home residents, and hospitalized patients. Obesity increases deficiency risk by 35% regardless of age or location. In the U.S., 50–60% of nursing home and hospital patients are deficient. Darker skin tones and extensive skin coverage, common in Middle Eastern countries, contribute to higher rates. Among infants,

47% of African American and 56% of Caucasian infants in the U.S. are deficient, compared to over 90% in Iran, Turkey, and India. In adults, 35% in the U.S. are deficient, versus over 80% in Pakistan, India, and Bangladesh. Among the elderly, deficiency rates are 61% in the U.S., and as high as 96% in India.

Pathophysiology:-

Vitamin D plays a crucial role in calcium homeostasis and bone metabolism. With chronic and/or severe vitamin D deficiency, a decline in intestinal calcium and phosphorus absorption leads to hypocalcemia, leading to secondary hyperparathyroidism. This secondary hyperparathyroidism then leads to phosphaturia and accelerated bone demineralization. This can further result in osteomalacia and osteoporosis in adults and osteomalacia and rickets in children.

Importance of Vitamin D:-

Vitamin D is an essential component for maintaining overall health, playing a pivotal role in regulating calcium balance within the blood and bones, crucial for bone formation and maintenance. Insufficient vitamin D results in compromised calcium and phosphorus absorption, leading to hypocalcemia and subsequent overactive parathyroid glands, causing secondary hyperparathyroidism.

Chronic vitamin D deficiency triggers symptoms like muscle weakness, cramps, fatigue, and depression. In an attempt to stabilize blood calcium levels, the body extracts calcium from bones, accelerating bone demineralization. This process can lead to osteomalacia in adults, characterized by soft bones, and rickets in children, causing bowed or bent bones due to ongoing growth.

The depletion of bone minerals heightens the risk of osteoporosis and osteomalacia-related fractures. In children, rickets impedes proper bone development. Understanding these intricate mechanisms is vital for researchers, shedding light on the far-reaching consequences of vitamin D deficiency and emphasizing the importance of maintaining optimal levels for overall skeletal health throughout life.^{ix}

Sources of Vitamin D :-

Vitamin D is obtained through sunlight, diet, and supplements. While skin synthesis via sun exposure is vital, excess exposure raises risks like skin aging and cancer, making diet and supplements important alternatives.

Diet plays a significant role in acquiring vitamin D, with certain foods like fatty fish, fortified dairy products, and egg yolks serving as dietary sources. Additionally, vitamin D supplements offer a convenient means of ensuring adequate intake, especially when natural sources may be limited.

Balancing sunlight, diet, and supplementation is key to optimizing vitamin D while avoiding excess sun exposure. A multifaceted approach ensures adequate levels, minimizes risks, and supports public health.

How much Vitamin D is needed :-

The amount of vitamin D you need each day depends on your age. The recommended amounts, in international units (IU), are:

Birth to 12 months: 400 IU

Children 1-13 years: 600 IU

Teens 14-18 years: 600 IU

Adults 19-70 years: 600 IU

Adults 71 years and older: 800 IU

Pregnant and breastfeeding women: 600 IU

People at high risk of vitamin D deficiency may need more. Check with your health care provider about how much you need.^x

What causes Vitamin D deficiency:-

In general, the two main causes of vitamin D deficiency are:

- Not getting enough vitamin D in your diet and/or through sunlight.
- Your body isn't properly absorbing or using vitamin D.
- There are several specific causes of vitamin D deficiency, including:

Certain medical conditions-

- Weight loss surgeries.
- Certain medications.

Several different biological and environmental factors can also put you at a greater risk of developing vitamin D deficiency, such as older age and the amount of melanin (pigment) in your skin.^{xi}

Medical conditions that can cause Vitamin D deficiency:-

Medical conditions that can cause vitamin D deficiency include:

- Cystic fibrosis, Crohn's disease and celiac disease: These conditions can prevent your intestines from adequately absorbing enough vitamin D through supplements, especially if the condition is untreated.
- Obesity: A body mass index greater than 30 is associated with lower vitamin D levels. Fat cells keep vitamin D isolated so that it's not released. Obesity often requires taking larger doses of vitamin D supplements to reach and maintain normal levels.
- In kidney or liver disease, reduced enzyme activity (hepatic 25-hydroxylase, renal 1-alpha-hydroxylase) impairs conversion of vitamin D to its active form, leading to deficiency.

Weight loss surgery and Vitamin D deficiency:-

Weight-loss surgeries that reduce the size of your stomach and/or bypass part of your small intestines, such as gastric bypass surgery, make it difficult for your body to absorb sufficient quantities of certain nutrients, vitamins and minerals.

If you've had weight-loss surgery, it's important to see your healthcare provider regularly so they can monitor your vitamin D levels and other nutrient levels. You'll likely need to take vitamin D supplements and other supplements throughout your life.^{xii}

Medications that can cause Vitamin D deficiency:-

Certain medications can lower vitamin D levels, including:

- Laxatives.
- Steroids (such as prednisone).
- Cholesterol-lowering drugs (such as cholestyramine and colestipol).
- Seizure-preventing drugs (such as phenobarbital and phenytoin).
- Rifampin (a tuberculosis drug).
- Orlistat (a weight-loss drug).

Always tell your healthcare provider about your medications and any supplements and/or herbs you take.^{xiii}

Symptoms and causes of Vitamin D deficiency:-

Lack of vitamin D isn't quite as obvious in adults. Signs and symptoms might include:

- Fatigue.
- Bone pain.
- Muscle weakness, muscle aches or muscle cramps.
- Mood changes, like depression.

However, you may have no signs or symptoms of vitamin D deficiency.^{xiv}

Treatment or Management of Vitamin D deficiency:-

Several preparations of vitamin D are available. Vitamin D3 (cholecalciferol), compared with vitamin D2 (ergocalciferol), has been shown to be more efficacious in achieving optimal 25-hydroxyvitamin D levels, thus favouring vitamin D3 as a treatment of choice.^{xv}

Prevention of Vitamin D Deficiency:-

Adults less than 65 years of age who do not have year-round effective sun exposure shall consume 600 to 800 international units of vitamin D3 daily to prevent deficiency. Older adults 65 years of age or more shall consume 800 to 1000 international units of vitamin D3 daily to prevent deficiency and reduce the risk of fractures and falls.

Management of Vitamin D Deficiency:-

The amount of vitamin D required to treat the deficiency depends largely on the degree of the deficiency and underlying risk factors.

- Initial supplementation for 8 weeks with Vitamin D3, either 6,000 IU daily or 50,000 IU weekly, can be considered.^{xvi} Once the serum 25-hydroxyvitamin D level exceeds 30 ng/mL, a daily maintenance dose of 1,000 to 2,000 IU is recommended.
- High-risk adults with vitamin D deficiency may require an initial dose of 10,000 IU/day of vitamin D3. Once serum 25-hydroxyvitamin D exceeds 30 ng/mL, a maintenance dose of 3,000–6,000 IU/day is advised.
- Vitamin D–deficient children need 2,000 IU/day or 50,000 IU weekly for 6 weeks, followed by 1,000 IU/day once serum 25(OH)D exceeds 30 ng/mL. The AAP advises 400 IU/day for breastfed infants and children consuming <1 L of fortified milk.
- Calcitriol can be considered where the deficiency persists despite treatment with vitamin D2 and/or D3. The serum calcium level shall be closely monitored in these individuals due to an increased risk of hypercalcemia secondary to calcitriol.
- Calcidiol can be considered in patients with fat malabsorption or severe liver disease.^{xvii}

Vitamin D deficiency in Adolescent Females:-

Vitamin D is essential for calcium absorption in the small intestine and proper bone mineralization. Deficiency reduces calcium absorption, leading to conditions like rickets in young children and poor bone health in adolescents. In adolescent girls, a group with unclear vitamin D status, deficiency may cause subclinical effects such as secondary hyperparathyroidism, low serum calcium, elevated alkaline phosphatase, and increased risk of bone abnormalities. The resurgence of rickets highlights the need for more research on vitamin D deficiency in adolescent girls, including its prevalence, risk factors, and prevention strategies.

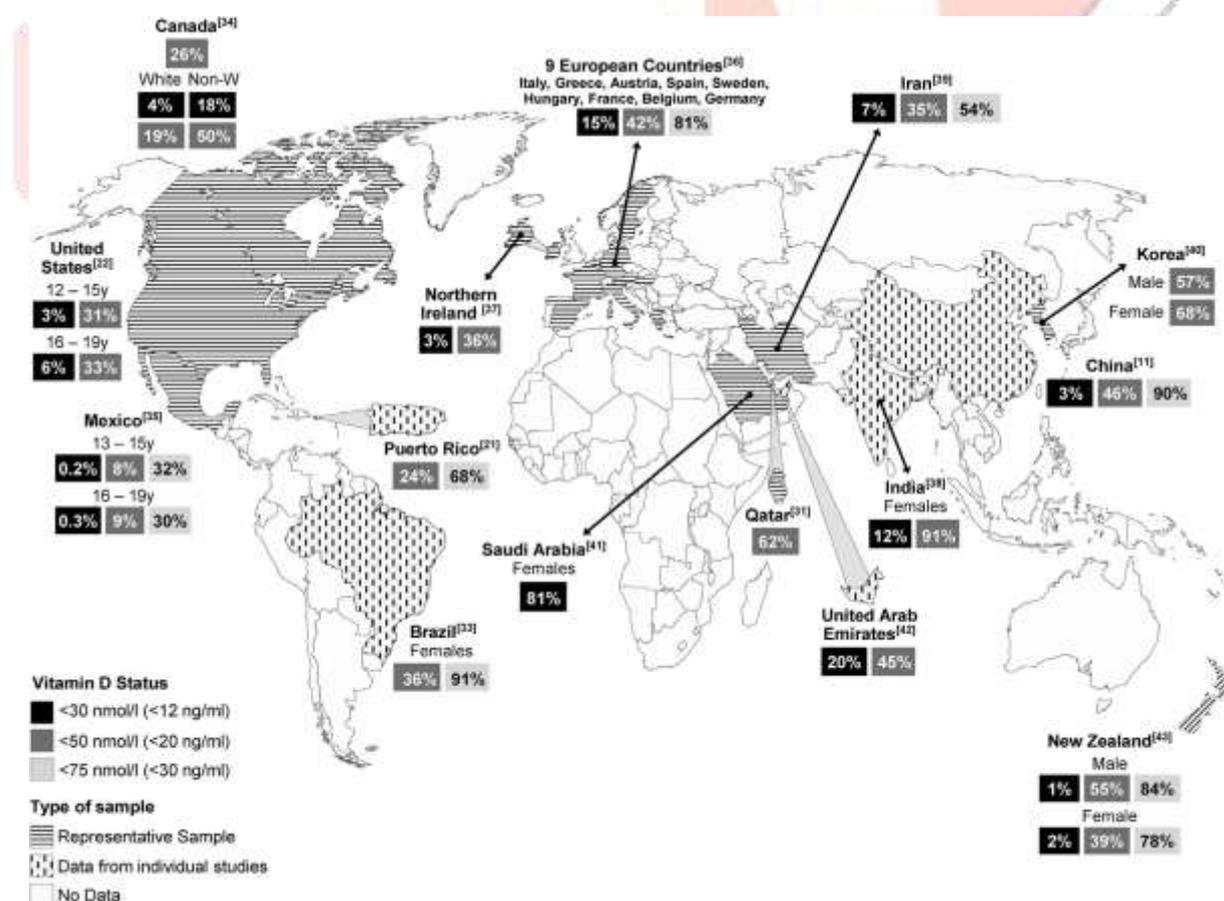


Fig:- Prevalence of low vitamin D status in adolescents worldwide.

Vitamin D deficiency in Reproductive age Women:-

The compelling evidence highlights the multifaceted impact of vitamin D deficiency on maternal and child health, prompting heightened public health concerns. Beyond adverse perinatal outcomes like hypertension, gestational diabetes, and miscarriages, deficiencies correlate with extra-skeletal issues such as osteoporosis, musculoskeletal pain, and an elevated fall risk. Negative perinatal consequences encompass impaired neonatal anthropometrics, a heightened risk of premature rupture of membranes, and infantile complications like eczema and nutritional rickets. Notably, the interplay of vitamin D deficiency and periodontal disease accentuates the association with preterm births and low birth weight, underscoring the imperative for comprehensive interventions and public policies at both primary care and national levels.^{xviii}

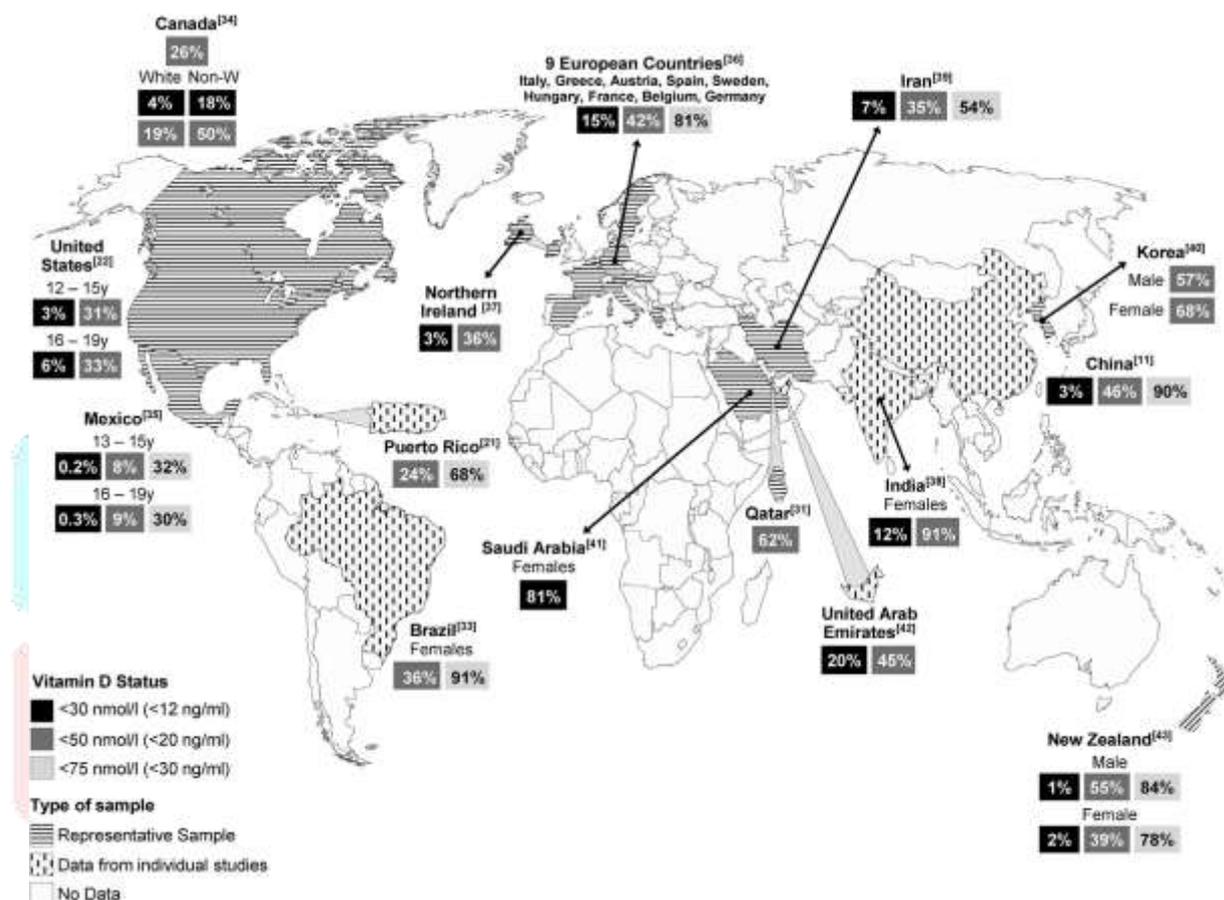


Fig:- Prevalence of low vitamin D status in adults worldwide.

Vitamin D deficiency in pregnant Women:-

Vitamin D is traditionally acknowledged for its vital role in bone health, particularly during pregnancy and childhood. Deficiency during intrauterine life and early childhood can result in severe outcomes such as rickets, growth retardation, skeletal deformities, and an increased fracture risk. While systematic Vitamin D supplementation sign for pregnant women was not universally recommended until recently, the shift in guidelines underscores its importance. Although there remains a lack of consensus on the optimal dosage for ensuring favourable pregnancy outcomes and childhood growth, most experts worldwide now advocate for Vitamin D supplementation during pregnancy. This reflects an evolving understanding of its critical role in maternal and child health.^{xix}

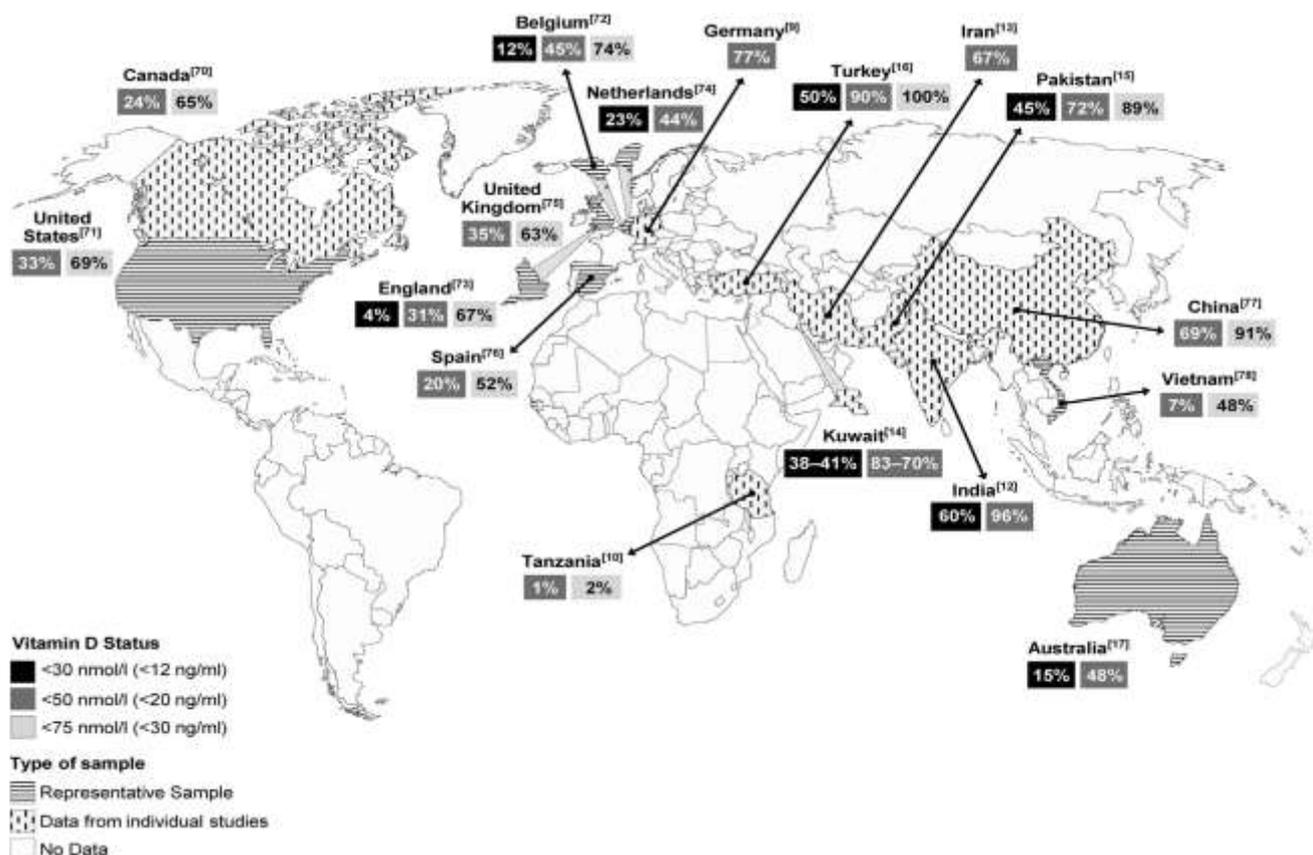
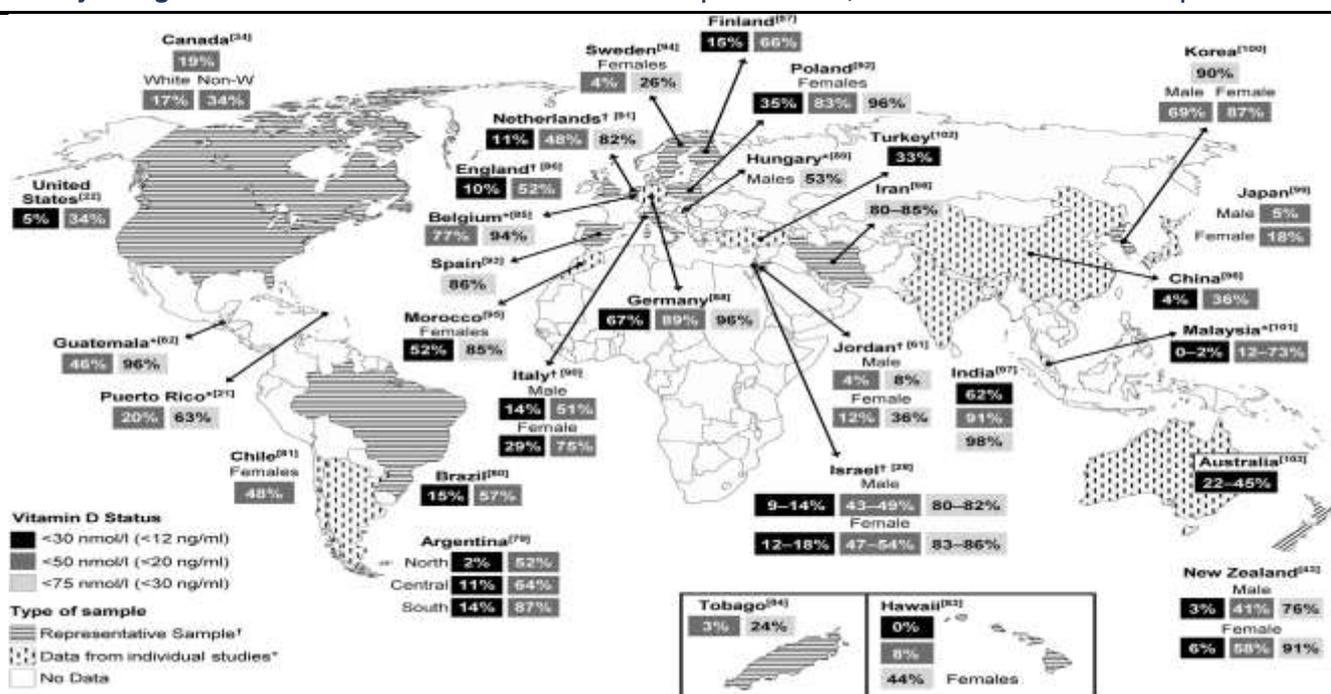


Fig:- Prevalence of low vitamin D status in pregnant or lactating women worldwide.

Vitamin D deficiency in Elderly Women:-

Osteoporosis, a prevalent skeletal disorder, is commonly linked to Vitamin D deficiency, particularly in the elderly. Vitamin D plays a crucial role in normal calcium and bone metabolism. While sunlight exposure is the primary source of Vitamin D, factors like latitude, season, and sunscreen use impact its cutaneous production. Elderly individuals face an elevated risk due to insufficient intake, limited sunlight exposure, reduced cutaneous and renal synthesis, and altered Vitamin D absorption. These factors result in diminished calcium absorption, prompting compensatory parathyroid hormone hypersecretion, leading to increased bone remodelling, cortical bone loss, and ultimately, osteoporosis. Addressing Vitamin D deficiency becomes crucial for preventing and managing this skeletal condition.^{xx}



Evaluation: -

High-risk individuals should be screened for vitamin D deficiency by measuring serum 25-hydroxyvitamin D. Optimal levels remain controversial, with racial differences in mineral metabolism complicating interpretation. For example, African Americans often have higher bone density and lower fracture risk despite lower vitamin D levels, and supplementation effects in non-White populations are not fully understood. The ISCD and IOF recommend maintaining serum levels ≥ 30 ng/mL in older adults to reduce falls and fractures. Levels >100 ng/mL may cause toxicity via hypercalcemia. In deficient patients, parathyroid hormone and serum calcium should also be assessed for secondary hyperparathyroidism.

Toxicity of Vitamin D:-

Vitamin D toxicity, though rare, occurs mainly from excessive oral intake rather than sun exposure, typically at serum 25-hydroxyvitamin D levels >88 ng/mL. Acute intoxication causes hypercalcemia, leading to confusion, anorexia, vomiting, polyuria, polydipsia, and muscle weakness. Chronic toxicity can result in nephrocalcinosis and bone pain. Research into its mechanisms highlights the need for a delicate balance in supplementation, with clear thresholds essential to maximize benefits while avoiding adverse effects.

Conclusion: -

The conclusion underscores the alarming prevalence of vitamin D deficiency in the Indian subcontinent, ranging from 70-100%. Dietary factors, including high fiber and phytate intake, are identified as contributors. To address this widespread issue, a collaborative effort between the public and private sectors is deemed crucial. The recommendation to revisit the cutoff levels for defining vitamin D deficiency in accordance with the needs of the Indian population suggests a potential mismatch with internationally set criteria. This highlights the importance of tailoring strategies to the specific context of India, cautioning against potential over-concern and emphasizing the necessity of a nuanced approach to combat vitamin D deficiency effectively.^{xxii}

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