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REVIEW ARTICLE

THE ROLE OF VITAMIN D IN HUMAN HEALTH: NARRATIVE REVIEW

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Vitamin D plays a vital development on the physiology of human beings, which is involved in various biological mechanisms. The most prominent of its roles is the control of skeletal health whereby it helps absorb calcium and phosphate thus guaranteeing proper bone growth and preventing diseases like rickets and osteoporosis.

In addition to its skeletal effects, vitamin D is necessary towards the regulation of immune response, cardiovascular, and cell growth as highlighted by several lists of experimental as well as epidemiological studies.

It is has become known that insufficiency of vitamin D is broadly associated to an increased number of chronic pathologies, such as cardiovascular disease, diabetes mellitus, or some malignancies, which highlight its role in the body system.

This review shows the multidimensional physiological functions of vitamin D, its endogenous and exogenous sources and highlights the consequences of deficiency hence the need to maintain optimal levels in the serum to promote healthy lifestyles.

Keywords: Vitamin D, human health, bone health, immune system, deficiency, chronic diseases, calcium absorption, supplementation

INTRODUCTION

The role of vitamin D in our body

Vitamin D is critical in a number of physiological activities, especially in the absorption of calcium, bones, immunomodulation as well as the inhibition of inflammatory pathways.

Lack of vitamin D seems to be one of the most widespread morbidities at a global level. Its consequences include impaired bone growth and strength, and increased predisposition to a variety of ubiquitous and life-threatening diseases, including different menaces, type 1 diabetes, cardiovascular diseases, and a host of autoimmune diseases [1,2].

The vitamin D is as well called the sunshine vitamin; that can be produced in the cutaneous tissues after exposure to ultraviolet radiation. The existence of this secosteroid is essential in maintenance of plasma calcium ion level in the physiological range of normalcy, thus, ensuring musculoskeletal health [3].

The American Geriatric Society, the National and International Osteoporosis, and Endocrine Society, characterize vitamin D insufficiency as a 25-hydroxyvitamin D (25 OH D) level below 30 ng/mL. The Endocrine Society supporters for an optimum limit of 40 to 60 ng/mL. The National Institute of Health describes vitamin D insufficiency as seems level as below 20 ng/ml. And some other sources categorize the inadequacy as sources 12 to 19 ng/mL and deficit as below 12 ng/mL [4,5].

The Endocrine Society, similarly, suggests a regular daily dose of 400 to 1000 International Units (IU) for the newborns below one year, 600 to 1000 IU for children and adolescents of around age 1 to 18 years, and 1500 to 2000 IU for most persons [6].

Inadequacy of vitamin D in children may lead to rickets and the inability of a child to achieve his maximum bone mass and height predetermined by genetic factors. Deficiency in vitamin D in the adult causes the irregular mineralization of collagen matrix in the child bone also called osteomalacia. Such collagen matrix is poor, lacks sufficient structural support and exposes one to risk of fracture. This excessive mineralized matrix can lead to periosteum that is seriously innervated system to progress

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outward and causes aching bones and is a frequent complaint with vitamin D deficient people. Muscle weakness and muscle pains are also caused by vitamin deficiency. Patients report about general bone and muscle pain. Vitamin D deficiency is present in about 40 -60 % of patients with generalized myalgias and bone pain [7]. Vitamin D deficiency (level < 30 ng/mL) and insufficiency (range 20 to 30 ng/mL) is an issue on a worldwide scale. Vitamin D deficiency is highly likely in pregnant women, people of African, Hispanic, obese, and children's populations. The lack of vitamin D is rampant in 50 percent of children below the age of 1-5 years and 70% below the age of 6-11 years. It is clearly stated that the rise in the number of obesity and reduced milk intake, and similar sun guard use under [8].

Mechanism of Action of vitamin D

Vitamin D is a hormone found from dietary supplements and produced through the skin. The wavelengths of ultraviolet B (UVB) radiation, differs from 290 to 315 nm, which is converted 7-dehydrocholesterol in the epidermis to nearly pre-vitamin D. Pre-vitamin D will undergo the process of heat isomerization to later turned to vitamin D, which is later turned into 25-hydroxyvitamin D (25 OH D) within the liver from both parts of the skin to synthesis and attain dietary sources; 25-hydroxyvitamin D will also serve as an essential indicator of vitamin D positions. 25-hydroxyvitamin D is later turned into its physiologically powerful form, 1,25-dihydroxyvitamin D (1,25 (OH)), within the kidneys via the enzymatic action of 25-hydroxyvitamin D-1 alpha-hydroxylase (CYP27B1). This powerful combination of parathyroid hormone, calcium, as well as phosphorus will control the renal source of 1,25-dihydroxyvitamin D [9].

The vitamin D receptor is a sort of hormone form of receptor that can be found within the nucleus and is found within the cell that is bound to 1,25-dihydroxy vitamin D. The most binding part of vitamin D on the vary receptor affects the gene transcript procedure and it enhances the expression of some genes and suppresses others. It may provoke the taking of calcium and phosphorus within the intestines. Irrespective of vitamin D, calcium as well as phosphorus within the diet are taken in non-absorbed quantities of almost 10 and 60-percent. With vitamin D, calcium and phosphorus are elevated with similar percentage of adjustment up to 30 percent to 40 percent and 80 percent, respectively.

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Vitamin D contains a physiologic function unlike the metabolism of calcium as indicated within the kidneys. Vitamin D receptor can be identified as the small intestine, colon, T and B lymphocytes, mononuclear cells, brain as well as the skin. The insulin can be provoked in synthesis, and regulate the action of the activated T and B lymphocytes, which can prevent inflammatory bowel diseases, and impacts the contractility of myocards [8,9].

Topical 1, 25-dihydroxy vitamin D can be applied in managing psoriasis. It can reduce erythema and the scaling of psoriasis. The part of the skin may have keratinocytes and eventually play a function in psoriasis and it can bear vitamin D receptors and like, vitamin D which can overturn the propagation of keratinocytes and tempts their diversity [10].

Importance and Prevalence of Vitamin D Deficiency:

There is no agreement on the optimum serum inductions of 25-hydroxyvitamin D; nevertheless, several experts categorize vitamin D insufficiency as a 25-hydroxyvitamin D level less than 20 ng per milliliter (50 nmol per liter) (11). The combination of 25-hydroxyvitamin D show a negative relationship with parathyroid hormone stages until the later achieves 30 to 40 ng per milliliter (75 to 100 nmol per liter), where parathyroid hormone levels start to raise at their minimum[12,13].

Furthermore, intestinal calcium transport within women can be supplemented by 45 to 65% when 25-hydroxyvitamin D levels can be raised from an average of 20 to 32 ng per milliliter (50 to 80 nmol per liter).Thirteen based on the data, a 25-hydroxyvitamin D level may differ from 21 to 29 ng per milliliter (52 to 72 nmol per liter) which determines related vitamin D deficiency while a near 30 ng per milliliter or a little higher shows enough vitamin D[14].Using the following values, it can be determined that almost a billion individuals universally are suffering from vitamin D inadequacy 7 to 12, 15 to 22 Abundant studies show that 40 to 100% of elderly men and women living in most communities in the U.S. and Europe are lacking vitamin D. in addition, above 50% of postmenopausal women experiencing treatment for osteoporosis show suboptimum levels of 25-hydroxyvitamin D, considered below 30 ng per milliliter (75 nmol per L).Children and adolescents similarly, may be at significant peril for vitamin D inadequacy[15]. For example, 52% of Hispanic and Black adolescents in a Boston study and 48% of white preadolescent girls in related Maine research showed 25-hydroxyvitamin D levels at lesser 20 ng

per milliliter. In similar studies, by the inference of winter, 42% of black girls as well as women of around 15 to 49 of age across the United States showed 25-hydroxyvitamin D levels at lesser than 20 ng per milliliter, and 32% of healthier students, patients and physicians, in a hospital in Boston, were identified to be lacking in vitamin D, despite the daily intake of a pure glass of milk, salmon, and a multivitamin for almost a week[16]. Similar project also took place in Europe. However, several individuals who reside near the equator and are facing insufficiency of sunlight protection may typically face high levels of 25-hydroxyvitamin D which exceeds 30 ng per milliliter[17-19]. Nevertheless, in most illuminated zones, vitamin D shortage is too much when the high number of the skin is covered from the sunlight[20]. Studies conducted within the region of Saudi Arabia, Australia, Turkey, United Arab Emirate Lebanon and India displayed that 30 to 50 % of children and adults showed 25-hydroxyvitamin D levels below 20 ng per milliliter.

Women with pregnancy or under lactation were seriously at risk as they were constantly thought to be under immunity to vitamin D insufficiency owing to their massive intake of daily prenatal multivitamin which contains 400 IU of vitamin D (70% consumed a prenatal vitamin, 90% took fish, and 93% swallowed an approximate 2.3 glasses of pure milk daily); however, 25-hydroxyvitamin D levels were less than 20 ng per milliliter in 73% of the women [21].

Evaluation of vitamin D level: High-risk individuals shall be assessed for vitamin D shortage.

Vitamin D shortage is assessed through the serum check 25-hydroxyvitamin D. Serum levels of 25-hydroxyvitamin D are still disputable as optimum [22]. Mineral absorption is very dissimilar across various races. One of such examples is the African Americans who are likely to experience higher bone density and lesser chance of fractures which is dissimilar from other races [23].

In addition, the possible inferences of the calcium and vitamin D addition in the non-white population do not fully evaluate and recorded. Minimum serum solutions of 25-hydroxyvitamin D of 30ng/ml are considered by the renowned International Society of Clinical Densitometry and the International Osteoporosis Foundation to decrease the risk of falls and fractures among older adults [24].

It lacks information on the higher limit of safe range of serum 25-hydroxyvitamin D but in high doses like above 100 ng/mL, toxicity is a possibility especially in second hypercalcemia. In a patient who has already been diagnosed with a deficiency in vitamin-D, secondary hyperparathyroidism should be screened, and the concentration of parathyroid hormone and serum calcium will be measured [24,25].

The etiology of vitamin D deficiency

Dietary sources and dermal synthesis like the fatty fish livers and stimulated foods, are the key contributors of and dietary sources, such as ergocalciferol (D2) and cholecalciferol (D3). These sorts of compounds can be later metabolized in the liver to 25-hydroxyvitamin D2 (25-OH-D2) and 25-hydroxyvitamin D3 (25-OH-D3) by the enzyme hepatic 25-hydroxylase (26). Thereafter, 25-OH-D2 and 25-OH-D3 are later turned into the biologically active sort of vitamin D,1,25-dihydroxyvitamin D, by the enzyme 1-alpha-hydroxylase within the kidneys. This form of active 1,25 dihydroxyvitamin D can improve intestinal calcium preoccupation and bone reabsorption whereas, reducing renal output of calcium and phosphate. Vitamin D inadequacy may go up from numerous factors [27].

Reduced dietary consumption and/or absorption

Malabsorption illnesses like the chronic pancreatic inadequacy, celiac disease, gastric bypass, short bowel syndrome, gastric bypass, inflammatory bowel disease, and cystic fibrosis can cause vitamin D deficiency. The reduced oral ingestion of vitamin D is more widespread among the population of high age [28].

Decreased sun exposure

Vitamin D synthesis by the cutaneous route decreases as age advances. The daily dose of sunshine needed to prevent deficiency is 20 minutes with more than 40% of the skin covered. These dark-skinned people have reduced cutaneous synthesis of vitamin D. Vitamin D shortage may also be caused by reduced contact to the sun through institutionalization of individuals or long hospitalization. Reduced efficacy of sun exposure is experienced among people who apply sunscreens regularly [29].

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Decreased endogenous synthesis

The malfunctioning of 25-hydroxylation may be observed in chronic liver disease like cirrhosis and the malfunction of the 1-alpha 25-hydroxylation in hyperparathyroidism, renal failure, and 1-alpha hydroxylase deficiency [29,28].

Increased hepatic catabolism

Phenobarbital, carbamazepine, dexamethasone, nifedipine, spironolactone, clotrimazole, and rifampin are some drugs that cause hepatic p450 enzymes to produce the vitamin D degradation effect. [30].

End organ resistance

End-organ resistance to vitamin D can be seen in hereditary vitamin D-resistant rickets.

Vitamin D Deficiency relation with cancer and Chronic Diseases
Cancer and vit D

Individuals in the higher latitudes are at risk of not only having Hodgkin lymphoma but also of cancer of the colon, cancer of the pancreas, prostate, ovaries, breast, and others and are more likely to succumb to these types of cancer, in comparison to those in lesser latitudes [31].
Prospective and retrospective epidemiological research tends to support that 25-hydroxyvitamin D below 20 ng/ml is linked to an amplified possibility of colon, prostate, and breast cancer as an augmented rate of mortality due to these malignancies [32,33].

A cohort examination of the Nurses' Health Study showed that the odds proportion for indicated colorectal cancer revealed a contrary link with the median serum levels of 25-hydroxyvitamin D (the odds proportion at 16.2 ng per milliliter [40.4 nmol per liter] was 1.0, whereas the odds proportion at 39.9 ng per milliliter [99.6 nmol per liter] was around 0.53; P≤0.01). Serum levels of 1,25-dihydroxyvitamin D were not in consonance with the colorectal cancer [34]. A potential study which examines vitamin D intake and colorectal cancer risk in men exposed a direct relationship, with a related risk of 1.0 for a consumption of around 6 to 94 IU/day and a relative risk of 0.53 for a consumption of 233 to 652 IU/day (P<0.05) [34]. Women were subjected to the maximum levels of sunlight during childhood and adolescence and displayed a 253% augmented the possibility of the development of colorectal cancer in the next eight years, in comparison to those with minimum sunlight contact, if their starting point showed 25-hydroxyvitamin D levels at lesser than 12 ng per milliliter (30 nmol per liter).A condensed risk of non-Hodgkin lymphoma was noticed in young adults before the illness begins alongside a significant likelihood of mortality from malignant melanoma developing in (35)(36)(37).

The paradox of this circumstance is that the production of is rigorously regulated by the kidneys.
Serum concentrations of 1,25-dihydroxyvitamin D do not raise with the increment of solar contact as well as vitamin D consumption. In addition, the vitamin D inadequacy, the level of 1,25-dihydroxyvitamin D is characteristically normal [38].
The possible rationale is that prostate, colon, and breast tissues prompt the enzyme 25-hydroxyvitamin D-1alpha-hydroxylase, thereby creating 1,25-dihydroxyvitamin D locally to control cancer-preventive genes that can sustain cell propagation and variation [39].
It has been suggested that in the presence of malignant cellular conditions, apoptosis may be induced by 1,25-dihydroxyvitamin D, while angiogenesis may be inhibited, hence reducing the likelihood of malignant cell survival [40].
Upon completion of its functions, 1,25-dihydroxyvitamin D initiates its degradation by activating the CYP24 gene to yield the inactive calcitroic acid. This will avoid 1,25-dihydroxyvitamin D from going into circulation to control calcium metabolism.

Autoimmune diseases, osteoarthritis and diabetes

Living at raised latitudes seems to be one of the risk factors that can intensify the possibility of emerging type 1 diabetes, multiple sclerosis, and Crohn's disease. Residing at less than 35 degrees latitude at the initial part, the case of multiple sclerosis will be like 50%. Among the white men and possibly women, the case of multiple sclerosis may reduce to around 41% with each increase of around 20 ng per milliliter in 25-hydroxyvitamin D directly above 24 ng per milliliter (60 nmol per liter) (odds ratio, 0.59; 95% CI, 0.36 to 0.97; P=0.04).71 Women who often took over 400 IU of vitamin D daily showed a 42% lessened chance of getting multiple sclerosis. Equivalent explanations have been observed for rheumatoid arthritis and osteoarthritis [41,42].
Several studies showed that the vitamin D addition in children may diminish the case of type 1 diabetes. To augment vitamin D intake in the process of gestation it may reduce the occurrence of islet autoantibodies in progeny [43]. In a group of 10,366 children found in Finland who established 2000 IU of vitamin D3 daily

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through their initial year of life and were observed for almost 31 years, this incidence of type 1 diabetes was later reduced by around 80% [44].
The peril among children with vitamin D inadequacy was raised by around 200% (of related risk, 3.0; 95% CI, 1.0 to 9.0). Another study showed that indicated that vitamin D inadequacy may raise insulin resistance, decreased insulin production and linked with metabolic syndrome, similarly, another 53 different studies showed that a daily intake of 1200 mg of calcium and 800 IU of vitamin D may reduce the danger of type 2 diabetes by 33% the risk of type 2 diabetes by 33% (and related risk, 0.67; 95% CI, 0.49 to 0.90) compared to a daily consumption of less than 400 IU of vitamin D [45].

Vit D relation with cardiovascular diseases

There seems to be a serious resistance to hypertension and cardiovascular disease due to the risk of staying at a very high latitude. A similar study of patients with hypertension showed to ultraviolet B radiation (3 times per week, for 3 months) displayed that the levels of vitamin D in the blood sample increased to an average 180% and the blood pressure reversed to normal (systolic and diastolic blood pressure reduced by almost 6mmHg).(46) Contact to ultraviolet B radiation also increased the levels of vitamin D within the blood by an average ot 180 and the blood pressure was recorded at normal range [47].

The severity of vitamin D deficiency

The sternness of vitamin D inadequacy is separated into:

(Mild, modest, and severe.)

Mild deficiency: 25-hydroxyvitamin D of lesser than 20 ng/mL

Moderate deficiency: 25-hydroxyvitamin D of lesser than 10 ng/mL

Severe deficiency: 25-hydroxyvitamin D of lesser than 5 ng/ml

The Regulation of Vitamin D

Vitamin D occurs in two main forms:

Vitamin D2 (ergocalciferol) and Vitamin D3 (cholecalciferol); the former is obtainable through diet mainly by fungi, but it could also be generated by plants and the latter may be generated by diet of animal products, or the turning of cholesterol +

precursor 7-dehydrocholesterol on contact to adequate ultraviolet B radiation. The fusion of the vitamin D might only be effective when the sun rays are at an angle of over 45° [48,49]. This has led to the fact that the northern hemisphere inhabitants lack adequate levels of vitamin D due to skin production in winter and in certain parts of the north, malfunctioning sun exposure can be up to 6 months of the year [50,51].

Also the average Western diet is low in vitamin D To augment vitamin D consumption, the policy of fortifying milk goods and margarine with vitamin D has been implemented by some countries, and another resource to augment vitamin D creation is the application of light bulbs to achieve artificial UVB contact [52].
Vitamin D needed to be used and this is a two-step procedure; the first one is the liver and the next one is in the extra-renal tissues basically the kidneys. Cholecalciferol is rapidly hydroxylated by the enzyme 25-hydroxylase (25-hydroxyvitamin D (25(OH)D)) (a CYP450-dependent enzyme also called CYP2R1) within the liver [53].
Subnormal plasma calcium or phosphate ions can control the stage of parathyroid hormone (PTH) and fibroblast development factor 23 which can cause the active vitamin D to be the 1α-hydroxylated 25(OH)D within the kidney and more especially the mitochondria of proximal complex tubule cells through the 1-hydroxylase enzyme (CYP27B1)[54].

1α-hydroxylation may also be emerged in the superficial tissues, which include epithelial tissues, bone, brain, endothelium placenta, endocrine glands, liver, endothelium, and predominantly in immune cells. 1,25(OH)2D can subsequently hinder 1α-hydroxylase and activate the 24-hydroxylase enzyme, which may degrade 25(OH)D, to establish a negative response tool that will regulate active vitamin D concentrations. The 24-hydroxylation of 25(OH)D produces 24,25(OH)2D, the sedentary metabolite, which may together in combination with the saturation of vitamin D existence can protect vitamin D toxicity [55,56]. Though, the active vitamin D form is with the saturation of 1,25(OH)2D, standard blood tests assess 25(OH)D as a result of storage of vitamin D. Conversely, the use of 1,25(OH)2D within the blood by the concentrations of PTH, calcium, and phosphates [57].
required protein (VDBP) and PTH, should be assessed in the determination of Vitamin D status and regular response to vitamin D addition on clinical result [58,59].

Vitamin D deficiency management

There should be a number of vitamin D prepartions. Vitamin D3 (cholecalciferol), compared to vitamin D2 (ergocalciferol), has been identified to be more efficient in the attainment of optimum 25-hydroxyvitamin D levels which makes vitamin D3 the treatment of selection [60,61].

Prevention of Vitamin D Shortage

Most adults under the age of 65 years, who suffer from the efficient sun contact throughout a year will be receiving 600 to 800 units and portion of vitamin D3 dail to prevent shortage. Humans of around 65 years of age and upward will receive around 800 to 1000 international units of vitamin D3 daily to prevent the shortage and less risk of fractures and falls.

Management of Vitamin D deficit

The requisite quantity of vitamin D for addressing the deficit is primarily contingent upon the sternness of the inadequacy and related risk factors.

One may consider the initial treatment of vitamin D3 for good eight weeks with a dose of around 6,000 IU daily or, perhaps, 50,000 IU in a week. When the blood 25-hydroxyvitamin D ratio is around 30 ng/mL, in a day to maintain the regulated dose of 1,000 to 2,000 IU is suggested [62].

Early vitamin D3 addition of 10,000 IU in a day will be needed to maintain maximum-risk adults that have deficiency vitamin D (Hispanic, African Americans, obese, and those taking some regular drug, malabsorption syndrome). Maintenance of 3000-6000 IU /day is as well, advisable following serum levels of 25-hydroxyvitamin D are greater than 30ng/mL [63,64].

Vitamin D lacking children demand 2000 IU/day of vitamin D3 or 50000IU of vitamin D3 once within the week for over 6 weeks. perhaps the serum 25(OH)D may be greater than 30 ng/mL, the management of the treatment is like 1000 IU/day. The American Academy of Pediatrics also recommend 400 IU of vitamin D addition in breastfed infants as well as in children who consume less than 1 L of vitamin D-content milk.

Calcitriol may also be taken whenever the inadequacy is not maintained despite the use of vitamin D2 and D3. Relatively, these sorts of people shall be taking serum calcium because of the risk of hypercalcemia resulted by calcitriol that may be considered in patients with fat malabsorption or strict liver disease.

Vitamin D intoxication

Vitamin D is one of the fat-soluble vitamins, thus the toxicity can occur, but it is not recorded. Hypervitaminosis D may be caused by the oral over consumption rather than contact with sun. A 25-hydroxyvitamin D serum level of over 88 ng/mL has been recorded as one of the toxics. Severe intoxication may occur in acute hypercalcemia which may turn to confusion, vomiting anorexia, polydipsia, polyuria, and muscle weakness. The frequent inebriation may cause nephrocalcinosis and pains in the bone [65].

The intoxication of vitamin D is quite unusual, but it may be due to accidental intake of massive doses. At the dose of above 50,000 IU/day, the levels of 25-hydroxyvitamin D surpass 150 ng/ml (374 nmol/liter) and are associated with hypercalcemia and hyperphosphatemia [66].

Doses of almost 10,000 IU of vitamin D3 in a day for a period of up to 5 months do not encourage toxicity. Individuals with lingering granulomatous diseases show maximum sensitivity to blood 25-hydroxyvitamin D solutions to exceed 30 ng per milliliter because of macrophage synthesis of 1,25-dihydroxyvitamin D, which results to hypercalciuria and hypercalcemia.

In these potential patients, 25-hydroxyvitamin D levels should be maintained at about 20 to 30 ng per milliliter to prevent vitamin D inadequacy and other hyperparathyroidism [67].

Vitamin D conveyance can happen orally to be later synthesized by the skin through ultraviolet contact. A concentration blood of about occur 25-hydroxyvitamin D (25 OH D) of about 30 ng/ml (78 nmoL/L) is expected to sustain the physiological role of vitamin D. It is also suggested use 25-hydroxyvitamin D (25 OH D) as an indicator of vitamin D position because of its half-life of two weeks; in contrast, 1,25-dihydroxyvitamin D (1,25 (OH)), the biologically active form, contains a serum and should not be used for th evaluation of vitamin D position [68,69].

The dermal production of vitamin D is contingent upon factors that can affect the nimber of UVB light and pass through the skin, which results in the analysis ans absorption of vitamin D by melanin in the skin. UVB radiation may be disrupted by the changes of 7-dehydrocholesterol into vitamin D. Accordingly, several individuals with the maximum skin pigmentation may have lesser cutaneous

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synthesis of vitamin D and need an additional period for the production of vitamin D under UVB radiation. A sunscreen with a protection of sun factor with (SPF) of 8 may reduce output by over 95% whereas a sunscreen with an SPF of 15 decreases it by over 98% [70].

During winter, UVB light strikes at a more oblique angle, resulting in increased absorption by the ozone layer. Consequently, there is diminished UVB radiation on the skin. Consequently, vitamin D production diminishes throughout the winter months. Similarly, UVB sunlight that reaches the skin at latitudes over 37 degrees will diminish, resulting in a reduction of vitamin D synthesis. In the early morning and evening, UVB photons from the sun arrive at an oblique angle, resulting in minimal synthesis by the skin.

Vitamin D is lipophilic and is kept in a dipose tissue. In individuals susceptive to obesity, a greater ratio of vitamin D is sequestered in adipose tissue, which results in a reduced presence for biological roles. Eventually, obese persons may need higher quantities of vitamin D addition to maintain the adequate serum levels of the vitamin [71].

Natural sources of vitamin D seem to be restricted. These can be referred to fatty fish like the salmon, sardines and mackerel. Milk and orange juice (100 units per 8-ounce serving), and some quantities of breads and cereals can be supported with vitamin D. Vitamin D additions may serve as an efficient source of oral vitamin D and are found both at over-the-counter through prescription. Are provided in dosages of 1000 IU, 2000 IU, 5000 IU, and 50,000 IU, and the attained via prescription [72].

CONCLUSION

To sum up, vitamin D is a nutrient of paramount significance, which has a complex effect on human health. It is essential to the skeletal integrity, immunological activity, and alleviation of chronic disease pathology, which is well established. Maintaining adequate levels of vitamin D can be attained through cutaneous synthesis in the presence of sunlight, intake of dietary means of consumption of dietary sources of vitamin D and through adjunct supplement regimens. However, the lack of vitamin D continues to be a widespread public health issue because of spatial and cultural differences that limit the amount of sun exposure, as well as, lifestyle habits. Control and prevention of the levels of vitamin D by both clinical and social health programs are essential towards maximizing the health outcome and alleviating the morbidity of the diseases that are related to its deficiency. More studies need to be carried out to explore its potential in therapy with numerous health conditions and develop more specific guidelines in relation to its optimal levels.

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Conflict of Interest

None to declare.

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