International Journal of Dermatology, Venereology and Leprosy Sciences

E-ISSN: 2664-942X
P-ISSN: 2664-9411
www.dermatologypaper.com
Derma 2023; 6(2): 50-55
Received: 18-06-2023
Accepted: 23-07-2023

Raghdah Magdy Dagher
MBBCH, Resident, Dermatology of Venereology, Faculty of Medicine, Tanta University, Egypt

Shady Fikry Abdel Salaam
MD, Lecturer of Dermatology and Venereology, Faculty of Medicine, Tanta University, Egypt

Yomna Mazid El-Hamd Neinaa
[1] Assistant Professor of Dermatology and Venereology, Faculty of Medicine, Tanta University, Egypt
[2] Dermatopathology unite, Faculty of Medicine, Tanta University, Tanta, Egypt

Nagwa Mohammad Elwan
[1] Dermatology and Venereology Department, Faculty of Medicine, Tanta University, Tanta, Egypt
[2] MD, Professor of Dermatology and Venereology, Faculty of Medicine, Tanta University, Egypt

Corresponding Author:
Raghdah Magdy Dagher
MBBCH, Resident, Dermatology of Venereology, Faculty of Medicine, Tanta University, Egypt

Role of Vitamin D in keloid scars

Raghdah Dagher, Shady Fikry Abdel Salaam, Yomna MEH Neinaa and Nagwa M Elwan

DOI: https://doi.org/10.33545/26649411.2023.v6.i2a.154

Abstract
Vitamin D (Vit. D) Is a fat-soluble substance. Humans get it endogenously on exposure to ultraviolet radiation or exogenously via its intake in the diet. It has a known role in homeostasis of calcium, mineralization of bone, immunity modification, and cell differentiation.
There are two types of vitamin D: vitamin D2 (ergocalciferol) that is found in plants and yeasts, and vitamin D3 (cholecalciferol) that may be found in animal sources or multivitamins. However, ultraviolet (UV) radiation from the sun causes the molecules that serve as precursors in the skin to take in vitamin D3, which accounts for 80–90% of the body's storage, then it will be hydroxylated in the liver to 25-hydroxyvitamin D and further hydroxylation will occur in the kidney in order to form 1,25-dihydroxyvitamin D. At the end, Vit. D is catabolized into calcitrioic acid in liver and excreted by the kidney.
A wide range of potential action is postulated by Vit. D upon various dermatological diseases including verrucae, hair disorders, acne, atopic dermatitis, skin cancers, vitiligo, psoriasis, cutaneous tuberculosis, ichthyosis, chronic urticarial, and keloid.
In keloid scars, Vit. D has been shown to prevent tissue fibrosis, inhibiting growth of keloid fibroblasts via preventing TGF -β induced extracellular matrix synthesis and serving as an anti-inflammatory agent.

Keywords: Keloid, Vitamin D, ultraviolet radiation

Introduction
Vitamin D (Vit. D) Is a fat-soluble compound [1]. The major form of Vit. D is 25-hydroxyvitamin D [25 (OH) Vit. D]. Vit. D is generated in the epidermis photochemically from the provitamin D, [7– dehydrocholesterol], via sunlight or artificial ultraviolet light action [2].
In addition to that, naturally, only little types of foods have Vit. D, such as milk, egg yolk, oily fish, oils from some fish livers, such as tuna and cod. Also, yeast and mushrooms have ergosterol that is provitamin D, which on UVB irradiation is transformed into Vit. D2. Also, vitamin D can be obtained through supplementation [2].

Metabolism of Vitamin D [3]
There are two types of vitamin D: vitamin D2 (ergocalciferol) that is found in plants and yeasts, and vitamin D3 (cholecalciferol) that may be found in animal sources or multivitamins. However, ultraviolet (UV) radiation of the precursor molecule 7-dehydrocholesterol within the skin is the principal supply of vitamin D3 (80–90% of the body storage).
Vitamin D attaches to the vitamin D binding protein (DBP) in the circulating blood stream and is carried to the hepatocytes, where it is hydroxylated to 25 hydroxy-vitamin D. This process occurs following endogenous production and isomerisation to vitamin D3 in the epidermal basal-layers or dietary consumption of vitamin D2/D3. The parathyroid hormone (PTH) stimulates the synthesis of the active secosteroid 1, 25 di-hydroxy vitamin D following 1α hydroxylation in the renal cells, whereas fibroblast growth factor 23 (FGF 23) and 1, 25 di-hydroxy vitamin D itself inhibit it. Finally, Vit. D is catabolized into calcitrioic acid in liver and excreted by the kidney, (Figure 1) [4].

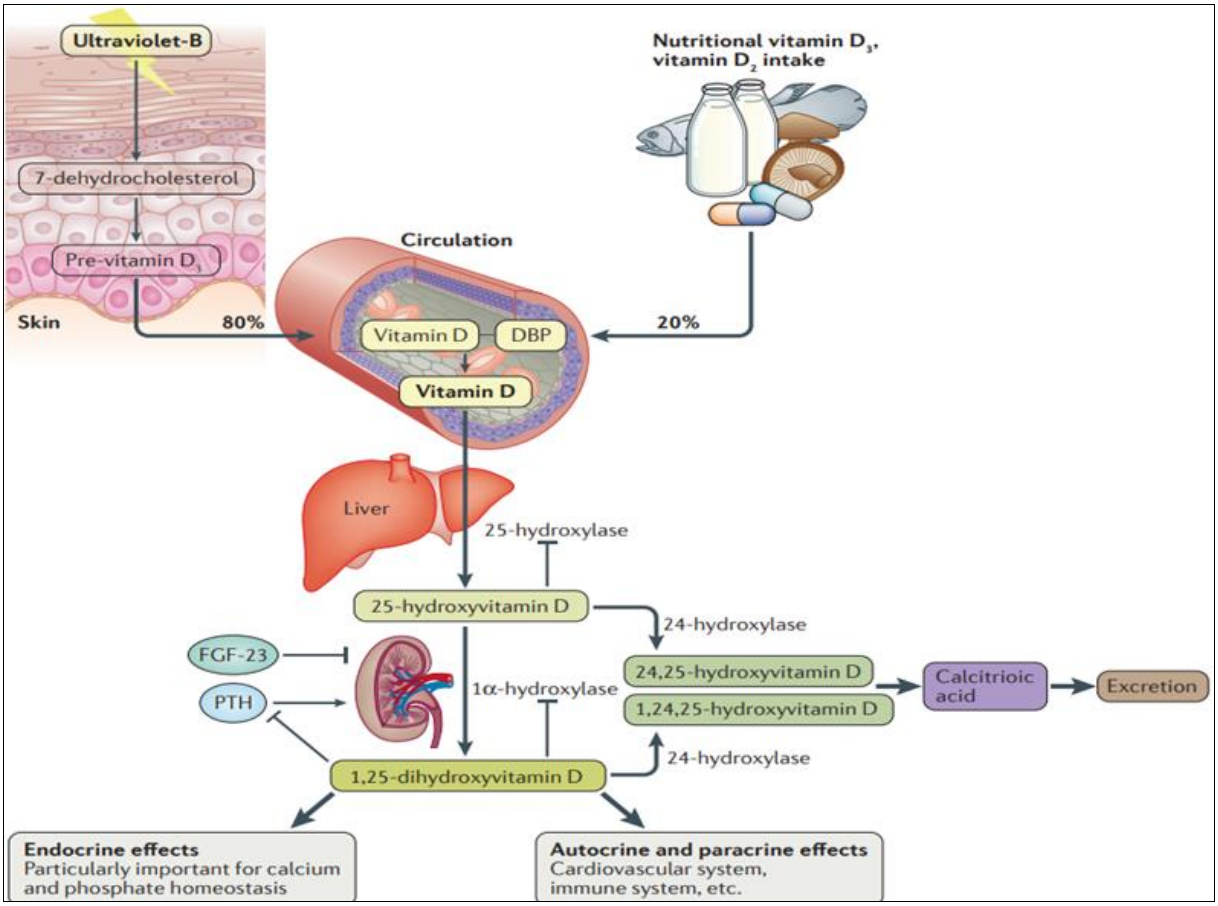

Fig. 1: Human metabolism of vitamin D [4].

The molecular mechanism of action of Vitamin D

The 1, 25-di-hydroxy Vit. D molecule attaches specifically to the vitamin D receptor (VDR) that works as a heterodimer with the retinoid X receptor (RXR) to activate target genes of Vit. D. When the heterodimeric complex of 1, 25(OH) 2D –VDR–RXR is just formed, it will interact with Vit. D response elements [a specific DNA sequences in and around target genes] leading to either transcription repression or activation [5].

Functions of Vitamin D

A wide range of potential functions is postulated by Vit. D:

I. Calcium homeostasis

The 1,25(OH)2D functions to elevate concentrations of calcium in the serum via three separate activities involving increase intestinal absorption, urinary reabsorption and finally mobilisation of calcium from the bone [6].

II. Immunity

Expression of VDR occurred in most of the cells of the immune system, including antigen-presenting cells (APC). In addition, macrophages can sometimes produce the enzyme of 25 OH Vit. D-1- hydroxylase that activates 25 OH Vit. D [7].

It can regulate the activity of T cells whether indirectly or directly via APC function modulation. It inhibits maturation of dendritic cells. Vit. D reduces expression of the cytokine Interleukin 12- (IL-12), which is necessary for maturation of T helper 1 cells [7].

Additionally, Vitamin D strongly stimulates T1/ST2 expression, a member of the IL-1 receptor families which is critical for normal T helper 2 (Th2) cell differentiation. Vit. D increased the producing of IL-4 in Th2 cells. The efficiency of 1, 25(OH) 2D in autoimmune diseases suppression had been found to depend on IL-2 and IL-4 [7].

Vitamin D also affects B cells directly and inhibits the production of immunoglobulins. Furthermore, B lymphocytes differentiation was found to be interrupted when exposed to 1, 25(OH) 2D *in vitro*. It could up-regulate the anti-microbial peptides production by keratinocytes, macrophages, and neutrophils. Furthermore, VDRE was found on the promoter of genes coding the anti-microbial peptides β-defensin and cathelicidin [7].

III. Cells differentiation

Vitamin D is important for proliferation of rapidly dividing cells which is essential for wound healing and growth. Also, it is important for cellular differentiation which leads to cells specialization for specific functions. Generally, cells differentiation results in a reduce in proliferation of cells. The 1, 25(OH) 2D, leads to inhibition of proliferation of cells and induction of differentiation [8].

IV. Regulation of blood pressure

The renin-angiotensin system (RAS) has long been known as a significant regulator of blood pressure and balance of electrolytes in mammals. Vit. D and the VDR have indirect effects on renin, mediated by the metabolism of calcium or PTH, both of them are recognized to independently affect expression of renin, (Figure 2) [4].

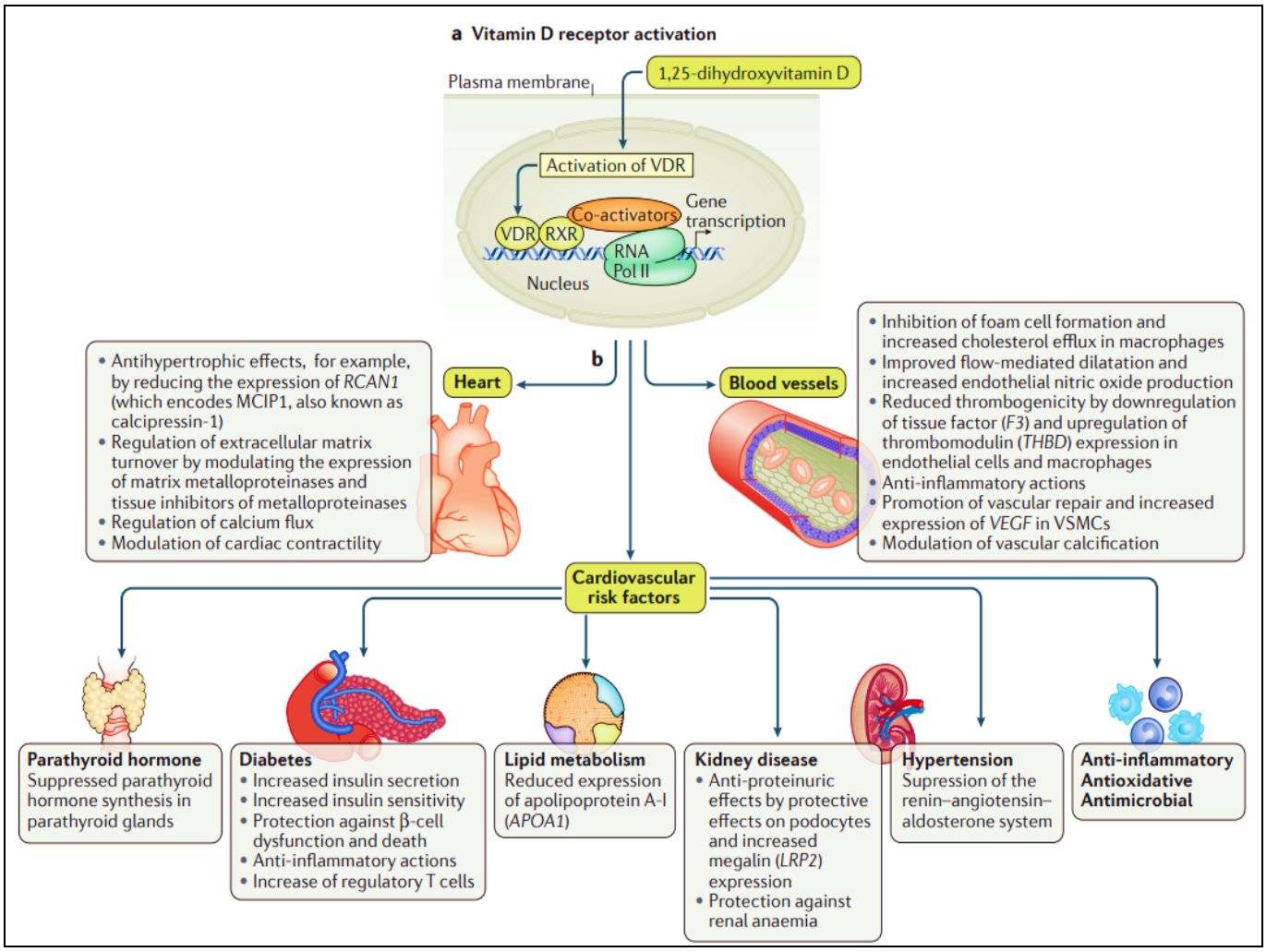

Fig 2: Cardiovascular effects of vitamin D receptor activation [4].

Vitamin D deficiency:

The level of 25-hydroxyvitamin D (25(OH) D) less than 20 ng/ml is the standard by which most professionals classify vitamin D insufficiency. When its level reached 30 ng/ml or below, a substantial reduce was existed in intestinal absorption of calcium that leads to raised PTH. PTH stimulates tubular calcium reabsorption and induces the kidneys to generate 1, 25 (OH) 2D. It also leads to activation of mature osteoclasts which dissolve the mineralized collagen matrix in bone, resulting in osteomalacia in adults and rickets in children. Secondary hyperparathyroidism results from the parathyroid glands being fully stimulated since vitamin D insufficiency worsens [9], (Figure 3) [10].

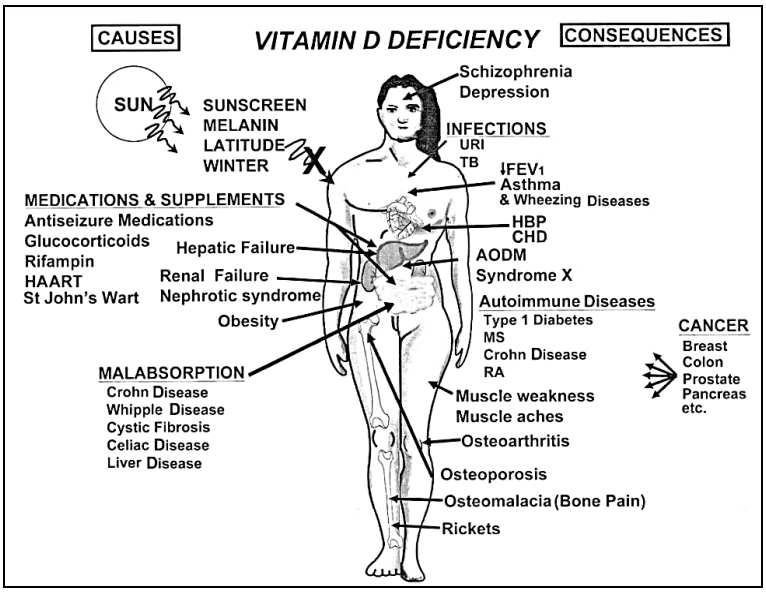

Fig 3: Causes and symptoms of vitamin D deficiency [10].

***URI:** Upper respiratory tract infection.
***TB:** Tuberculosis.
***FEV1:** Forced expiratory volume
***HBP:** High blood pressure.
***CHD:** Coronary heart disease.
***AODM:** Adult-onset diabetes mellitus.
***MS:** Multiple sclerosis.
***RA:** Rheumatoid arthritis.
***HAART:** Highly active antiretroviral therapy.

Vitamin D and keloid

By controlling apoptosis-related genes, 1, 25 (OH) 2D suppresses mesenchymal multi-potent cells and primary pulmonary fibroblasts. Additionally, it induces an anti-fibrotic phenotype and causes the activation of other important ant fibrotic factors [11]. Additionally, it was discovered that no relationship was existed among serum 25(OH) D levels and either age, gender, period of keloid, or familial history, and the more extreme the degree of keloid, the reduced level of serum 25-hydroxyvitamin D [11-13].

Keloid patients are more prone to have VD Deficiency (VDD), hypertension, and dark skin. Dark-skinned people are also more likely to have VDD and hypertension. Sunlight exposure is the major source of VD, with skin pigmentation being a crucial driver in lower UVB penetration, resulting in decreased cutaneous Vit. D3 production. Individuals with very dark skin, such as those found in some African communities [14].

Keloid fibroblasts show a dose-dependent reduction in proliferation in response to Vit. D3 therapy, with collagen I expression dropping thrice in the treated samples, indicating that, Vit. D3 has an effect on keloid regression. TGF-β1/Smad signaling is negatively regulated by the vitamin D receptor. In patients with systemic sclerosis, VDR suppression increases fibroblast sensitivity to TGF-β1, and activation of VDR with paricalcitol lowers TGF-β1's stimulatory action on fibroblasts, inhibiting collagen synthesis and myofibroblast differentiation [14].

In addition, paricalcitol promotes the creation of VDR and phosphorylated Smad3 complexes, which inhibits Smad transcriptional activity, which controls the production of profibrotic genes such as MMPs, proteoglycans, integrin, and plasminogen activator. The susceptibility genes for keloid have been identified as plasminogen activator and VDR [14].

VDR expression is considerably reduced in keloid patient's peripheral blood cells, epidermis, and fibroblasts from patients with systemic sclerosis, another fibrotic illness [14].

Owing to the keloid scars' large form, ongoing development, and challenging treatment, managing keloid scars continues to be a tough task for dermatologists [15]. Although intralesional injections of corticosteroid is the first-line method of treating keloids, recent research conducted by Mamdouh *et al*., 2022 [14] evaluated the possible use of intralesional vitamin D injection as a distinct beneficial reliable, and affordable approach for treating keloid scars. Based on a statistically highly significant decrease in VSS, the researchers of this study, which included 40 patients with keloid lesions, concluded that weekly intralesional injections of vitamin D3 (given over the course of three to four sessions) were effective in healing keloid scars. They came to conclude that vitamin D could minimize the risk of recurrence, enhance that proliferative scar becoming visible, and solve the drawbacks of traditional keloid therapy approaches that includes cryo-therapy, intra-lesional 5-FU, and intra-lesional corticosteroids. They explained their results by the role of Vit. D in avoiding tissue fibrosis, inhibiting growth of keloid fibroblasts via preventing TGF - β induced extracellular matrix synthesis and serving as an anti-inflammatory agent.

Vitamin D and dermatological diseases

Vitamin D and verrucae: It has been discovered that vitamin D directly influences the control of innate immune responses to the human papilloma virus. Anti-microbial peptides (AMPs), such as the β- and α-defensins and cathelicidins, undergo expression via reactions to 1, 25(OH) 2D on human keratinocytes, are a key part of this system, neutrophils, and monocytes and has many functions including chemotaxis, cytokine, and chemokine production [16]. Other possible suggested mechanisms include keratinocytes differentiation and apoptosis, inhibition of hyperkeratosis and also immunomodulation [17].

Vitamin D and hair

The late anagen and catagen phases of the hair cycle are when VDR expression is highest in the hair follicles. Therefore, it must be expressed for the natural hair cycle to continue. Additionally, it was recently shown that a deficiency of VDRs inhibits the formation of hair follicles and epidermal differentiation. It was reported that treatment by Vit. D analogues leads to qualitative and accelerated improving of the hair regrowth but doesn't prevent alopecia induced by chemotherapy [18].

Role of vitamin D in acne

Intake of Vit. D orally was found to have therapeutic value in acne via its comedolytic and antioxidant properties [19]. Cutaneous metabolism or synthesis of Vit. D metabolites might be significant for regulation of both growth and sebum production [20].

Vitamin D and atopic dermatitis

According to epidemiological research, atopic dermatitis sufferers consume less vitamin D than healthy controls. [9-21].

Vitamin D and skin cancer

Vitamin D was found to be involved in growth regulation and epidermal cells differentiation as well as immunomodulating effects and chemoprevention of skin cancers induced by UVR [22]. Human skin cells were protected from cell death and apoptosis induced by UV radiation by 1,25 (OH)2D [23].

Genetic VDR polymorphisms were reported to have increased risk of development of cutaneous malignant melanoma [24]. In additions, it was reported that the VDR expression is markedly elevated in tumor cells of basal and squamous cell carcinomas in comparison to adjacent epidermis of unaffected tissues [25].

Vitamin D was found to have antiproliferative and differentiating effects on malignant and non-malignant cells via stimulating the expression of p21, p27 [26].

Vitamin D and vitiligo

Vitamin D may impose its impacts of immunomodulation by inhibition of TNF-α, TNF-γ, IL-6, and IL-8 expression which have a role in vitiligo pathogenesis [27]. It was also

demonstrated that the Apa-I polymorphism of the VDR gene is correlated to vitiligo [28]. Previous studies reported that, Vit. D levels were very low or insufficient in most patients complaining of vitiligo vulgaris. Hence, Vit. D supplementation could be utilized in the management of autoimmune diseases like vitiligo [29, 30].

Vitamin D and psoriasis

About 60 years ago, oral Vit. D supplements was first reported as a therapeutic option for psoriasis depending on the sunlight favourable impacts on the illness. However, oral form is now limited because of its side effects [17]. Unlike oral Vit. D, topical Vit. D analog (calcipotriol) is used effectively and safely in psoriasis therapy without its systemic effects [31].

Vitamin D inhibits the generation of (IFN)-γ, IL-2 and IL-6 that are strong mediators of inflammation [32]. It induces activity of T-suppressor cells and inhibits formation of natural killer and cytotoxic cells [17]. Another possible mechanism involves normalization of distribution and expression of integrin namely, intercellular Adhesion Molecule-1 (ICAM-1), HLA DR, and CD26 along the dermal-epidermal junction [33].

Vitamin D and cutaneous tuberculosis

Since the 1940s, calciferol has been used for treating lupus vulgaris. Without being very toxic, calciferol had a fantastic therapeutic impact on lupus vulgaris. By means of systematic review and meta-analysis, several researchers came to the conclusion that low blood vitamin D concentrations are linked to a greater risk of active TB [21].

Vitamin D and ichthyosis

In the literature, multiple instances of a connection between rickets and keratinization abnormalities were existed. The proliferation of keratinocytes is inhibited by calcitriol. As a result, conditions like ichthyosis, which is brought on by aberrant keratinization, may be linked to changes in vitamin D metabolism resulting in rickets and osteomalacia. Although it is still uncertain whether this link is coincidence or causative [21].

Even though oral vitamin D supplementation for individuals with coexisting pathologies restored biochemical values and healed rickets, substantial improvements in ichthyosis has not been shown despite research showing a link between deficiencies in vitamin D and keratinization conditions [34].

Vitamin D and systemic lupus erythematosus (SLE)

A systemic inflammatory illness, systemic lupus erythematosus is influenced by environmental, inherited, hormonal, and immunological variables. Insufficiency in vitamin D is one of these reasons. Low blood vitamin D concentrations among individuals with SLE seem to interfere with the clinical aspects of the disease's activity and chronicity [35].

Vitamin D and systemic sclerosis (SSC)

While *in vitro* research indicates that vitamin D may have antifibrotic properties that might be useful in the treatment of SSc, clinical evidence supporting this discovery *in vivo* is presently insufficient. However, people with SSc often have vitamin D insufficiency [36].

Vitamin D and polymorphic light eruption (PLE)

An idiopathic photosensitivity condition called polymorphic light eruption is predominantly caused by ultraviolet (UV) radiation exposures. When vitamin D concentrations were raised with phototherapy using UV-B (311 nm), Gruber-Wackernagel *et al*. [37] reported that individuals with PLE had fewer levels than controls and that there was a link among increased levels and clinical enhancement of the lesions. Additionally, topical use of calcipotriol for a week prior to exposure decreased symptoms in almost one-third of the individuals exposed to photo-provocation and was recommended as a potential preventative strategy.

Vitamin D and hidradenitis suppurativa (HS)

Hidradenitis suppurativa is an inflammatory skin condition that affects people's quality of life significantly. There has been research on the association among HS and vitamin D levels, showing that individuals with HS had fewer levels of vitamin D compared to controls and that there was an inverse relationship among vitamin D concentrations and disease intensity. 79% of the participants observed improvement in their inflammatory nodules after taking vitamin D supplements for a six-month period [38].

Vitamin D and Chronic urticaria

Reduced serum concentrations of vitamin D were stated in individuals with chronic spontaneous urticaria contrasted with healthy controls, and vitamin D supplementation in such patients can be used as adjuvant therapy. Severe vitamin D deficiency could trigger the progression of acute urticaria to chronic urticaria and autologous serum skin test-positive patients had even lower serum vitamin D levels. The exact mode of action of vitamin D in urticaria is unclear. Proposed immunological roles include suppression of IL-1, IL-6, IL-12, IFN-γ, and normal T cell expression and secretion by vitamin D [39].

Conflict of Interest

Not available

Financial Support

Not available

References

1. Ng SC, Cheng TS. Vitamin D and dermatology. Hong Kong J Dermatol Venereol. 2014;22:124-9.
2. Dominguez LJ, Farruggia M, Veronese N, Barbagallo M. Vitamin D sources, metabolism, and deficiency: available compounds and guidelines for its treatment. Metabolites. 2021;11(4):255-87.
3. Saponaro F, Saba A, Zucchi R. An update on vitamin D metabolism. Int J Mol Sci. 2020;21(18):6573-91.
4. Pilz S, Verheyen N, Grübler MR, Tomaschitz A, März W. Vitamin D and cardiovascular disease prevention. Nat Rev Cardiol. 2016;13(7):404-17.
5. Bikle DD, Adams JS, Christakos S. Vitamin D: production, metabolism, mechanism of action, and clinical requirements. In: Rosen CJ, Compston JE, Lian JB (eds.) Primer on the metabolic bone diseases and disorders of mineral metabolism. 7th edn. Washington: American Society for Bone and Mineral Research; 2009;28:141-9.
6. DeLuca HF. Overview of general physiologic features and functions of vitamin D. Am J Clin Nutr. 2004;80(6):1689s-96.
7. Hewison M. An update on vitamin D and human

immunity. Clin Endocrinol (Oxf). 2012;76(3):315-25.
8. Holick MF. Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease, and osteoporosis. Am J Clin Nutr. 2004;79(3):362-71.
9. Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266-81.
10. Holick MF, Chen TC. Vitamin D deficiency: A worldwide problem with health consequences. Am J Clin Nutr. 2008;87(4):1080S-6S.
11. Ramirez AM, Wongtrakool C, Welch T, Steinmeyer A, Zügel U, Roman J. Vitamin D inhibition of pro-fibrotic effects of transforming growth factor beta1 in lung fibroblasts and epithelial cells. J Steroid Biochem Mol Biol. 2010;118(3):142-50.
12. Damanik VI, Putra IB, Ginting O. Correlation between serum 25-hydroxyvitamin D levels with keloid severity. Open Access Maced J Med Sci. 2019;7(1):65-7.
13. Lee DE, Trowbridge RM, Ayoub NT, Agrawal DK. High-mobility group box protein-1, matrix metalloproteinases, and vitamin D in keloids and hypertrophic scars. Plast Reconstr Surg Glob Open. 2015;3(6):e425-e33.
14. Frankel C. Role of Vitamin D in Keloid Disorder. Plast Surg Nurs: Case Studies. 2022;3(1):1-2.
15. Mamdouh M, Omar GA, Hafiz HSA, Ali SM. Role of vitamin D in treatment of keloid. J Cosmet Dermatol. 2022;21(1):331-6.
16. Gombart AF, Borregaard N, Koeffler HP. Human cathelicidin antimicrobial peptide (CAMP) gene is a direct target of the vitamin D receptor and is strongly up-regulated in myeloid cells by 1,25-dihydroxyvitamin D3. FASEB J. 2005;19(9):1067-77.
17. Shahriari M, Kerr PE, Slade K, Grant-Kels JE. Vitamin D and the skin. Clin Dermatol. 2010;28(6):663-8.
18. Amor KT, Rashid RM, Mirmirani P. Does D matter? The role of vitamin D in hair disorders and hair follicle cycling. Dermatol Online J. 2010;16(2):3.
19. Bowe WP, Logan AC. Clinical implications of lipid peroxidation in acne vulgaris: old wine in new bottles. Lipids Health Dis. 2010;9:141.
20. Reichrath J, Schuler CH, Seifert M, Zouboulis CH, Tilgen W. The vitamin D endocrine system of human sebocytes. Exp Dermatol. 2006;15(8):643-8.
21. Wadhwa B, Relhan V, Goel K, Kochhar AM, Garg VK. Vitamin D and skin diseases: A review. Indian J Dermatol Venereol Leprol. 2015;81(4):344-55.
22. Lehmann B. Role of the vitamin D3 pathway in healthy and diseased skin-facts, contradictions and hypotheses. Exp Dermatol. 2009;18(2):97-108.
23. Reichrath J. Vitamin D and the skin: An ancient friend, revisited. Exp Dermatol. 2007;16(7):618-25.
24. Mocellin S, Nitti D. Vitamin D receptor polymorphisms and the risk of cutaneous melanoma: A systematic review and meta-analysis. Cancer. 2008;113(9):2398-407.
25. Kamradt J, Rafi L, Mitschele T, Meineke V, Gärtner BC, Wolfgang T, *et al*. Analysis of the vitamin D system in cutaneous malignancies. Recent Results Cancer Res. 2003;164:259-69.
26. Fleet JC, DeSmet M, Johnson R, Li Y. Vitamin D and cancer: A review of molecular mechanisms. Biochem J. 2012;441(1):61-76.
27. AlGhamdi K, Kumar A, Moussa N. The role of vitamin D in melanogenesis with an emphasis on vitiligo. Indian J Dermatol Venereol Leprol. 2013;79(6):750-8.
28. Birlea S, Birlea M, Cimponeriu D, Apostol P, Cosgarea R, Gavrila L, *et al*. Autoimmune diseases and vitamin D receptor Apa-I polymorphism are associated with vitiligo in a small inbred Romanian community. Acta Derm Venereol. 2006;86(3):209-14.
29. Ustun I, Seraslan G, Gokce C, Motor S, Can Y, Ugur Inan M, *et al*. Investigation of vitamin D levels in patients with vitiligo vulgaris. Acta Dermatovenerol Croat. 2014;22(2):110-3.
30. Silverberg JI, Silverberg AI, Malka E, Silverberg NB. A pilot study assessing the role of 25 hydroxy vitamin D levels in patients with vitiligo vulgaris. J Am Acad Dermatol. 2010;62(6):937-41.
31. Trémezaygues L, Reichrath J. Vitamin D analogs in the treatment of psoriasis: where are we standing and where will we be going? Dermatoendocrinol. 2011;3(3):180-6.
32. Azizieh F, Alyahya KO, Raghupathy R. Association between levels of vitamin D and inflammatory markers in healthy women. J Inflamm Res. 2016;9:51-7.
33. Soleymani T, Hung T, Soung J. The role of vitamin D in psoriasis: A review. Int J Dermatol. 2015;54(4):383-92.
34. Thacher TD, Fischer PR, Pettifor JM, Darmstadt GL. Nutritional rickets in ichthyosis and response to calcipotriene. Pediatrics. 2004;114(1):e119-23.
35. Schneider L, dos Santos ASP, Santos M, da Silva Chakr RM, Monticielo OA. Vitamin D and systemic lupus erythematosus: State of the art. Clin Rheumatol 2014;33(8):1033-8.
36. Perazzi M, Gallina E, Manfredi GF, Patrucco F, Acquaviva A, Colangelo D, *et al*. Vitamin D in systemic sclerosis: A review. Nutrients. 2022;14(19):3908-22.
37. Gruber-Wackernagel A, Bambach I, Legat FJ, Hofer A, Byrne SN, Quehenberger F, *et al*. Randomized double-blinded placebo-controlled intra-individual trial on topical treatment with a 1,25-dihydroxyvitamin D3 analogue in polymorphic light eruption. Br J Dermatol. 2011;165(1):152-63.
38. Navarro-Triviño FJ, Arias-Santiago S, Gilaberte-Calzada Y. Vitamin D and the skin: A review for dermatologists. Actas Dermo-Sifiliográficas (English Edition). 2019;110(4):262-72.
39. El Mongy N, Hilal R. Vitamin D and the skin: An immunologic and therapeutic update. J Egypt Women's Dermatologic Soc. 2023;20(2):69-80.

How to Cite This Article
Dagher R, Salaam SFA, MEH Neinaa Y, Nagwa M Elwan. Role of Vitamin D in keloid scars. International Journal of Dermatology, Venereology and Leprosy Sciences. 2023; 6(2): 50-55.

Creative Commons (CC) License
This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 International (CC BY-NC-SA 4.0) License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.