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VITAMIN D IN DERMATOLOGICAL DISEASES: MECHANISMS, CLINICAL EVIDENCE AND THERAPEUTIC PERSPECTIVES

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ABSTRACT

BACKGROUND

Vitamin D functions as a prohormone involved in cutaneous homeostasis. The skin is both the site of its synthesis and a target organ for its active metabolites, which influence keratinocyte proliferation, differentiation, and innate immune responses. Experimental and clinical literature suggests that vitamin D related pathways may be relevant to the pathogenesis and clinical course of several dermatological diseases.

AIM

To present the current state of knowledge regarding the role of vitamin D in dermatology, with particular emphasis on psoriasis, atopic dermatitis (AD), acne vulgaris, and skin cancers.

Methods: This article is a narrative review based on a qualitative synthesis of published literature identified through non systematic searches of PubMed, Scopus, and Google Scholar. The review primarily considered publications from 2012 to 2025, with selective inclusion of earlier key studies when necessary to describe fundamental mechanisms of vitamin D synthesis, metabolism, and action in the skin. Included sources comprised experimental studies, clinical observational studies, randomized and non randomized clinical trials, narrative reviews, and meta analyses relevant to vitamin D metabolism, skin physiology, and the selected dermatoses.

RESULTS

Vitamin D is involved in epidermal differentiation, immune modulation, and antimicrobial peptide production. In psoriasis, topical vitamin D analogues have an established therapeutic role, while studies assessing serum 25(OH)D levels and their association with disease severity report inconsistent findings. In AD, vitamin D is discussed in relation to barrier integrity, antimicrobial defense, and immune regulation, but clinical evidence for supplementation effects

remains heterogeneous. In acne vulgaris, mechanistic studies suggest anti inflammatory effects and inhibition of sebocyte proliferation, whereas clinical data are limited. With respect to skin cancer, current evidence does not confirm a protective role of vitamin D in prevention.

CONCLUSIONS

Vitamin D has a multifaceted role in skin physiology, but the strength of clinical evidence varies across diseases. Evidence is strongest for its therapeutic use in psoriasis through topical analogues. For AD and acne, available data remain largely observational or mechanistic and do not justify routine therapeutic recommendations. Assessment of serum 25(OH)D levels and correction of confirmed deficiency may be considered as part of general medical care, but should not replace standard dermatological treatment. Further well designed prospective and randomized studies with clearly defined dermatological endpoints are needed to clarify the clinical relevance of vitamin D beyond psoriasis.

Keywords: vitamin D; psoriasis; atopic dermatitis; acne; skin cancer; immune modulation; epidermal differentiation

INTRODUCTION

Vitamin D_3 chemically 1α,25-dihydroxycholecalciferol is a steroid organic compound commonly classified as a vitamin. It should be emphasized that it also functions as a hormonal component [1,3]. Its precursor is cholesterol supplied to the human body exogenously through the diet and endogenously via biosynthesis [1]. The skin plays a crucial role in the synthesis of this vitamin. In the basal layer of the epidermis cholesterol is converted into provitamin D_3 7 dehydrocholesterol which under ultraviolet B radiation undergoes a photochemical transformation to form previtamin D_3. In the subsequent step isomerization occurs resulting among other compounds in cholecalciferol traditionally referred to as vitamin D_3 [2]. It is estimated that cutaneous biosynthesis of cholecalciferol accounts for as much as 80 to 100 percent of the body's vitamin D requirements [2]. Physiologically vitamin D in humans is primarily associated with calcium phosphate homeostasis within the musculoskeletal system yet the biological properties of this compound are far broader [3]. Focusing specifically on human skin it not only participates in vitamin D synthesis but also serves as a target organ for its active form [4,7]. The vitamin D receptor is present in keratinocytes fibroblasts Langerhans cells and T and B lymphocytes indicating that its activity encompasses numerous dermatological processes [4,7,39]. In the skin vitamin D signaling is mechanistically linked to keratinocyte proliferation and differentiation immune modulation and induction of antimicrobial peptides which together support barrier integrity and cutaneous innate defense [4,7,8]. At the clinical level these pathways are relevant to chronic inflammatory dermatoses and have been most clearly translated into practice through topical vitamin D analogues in psoriasis while associations between serum 25 hydroxyvitamin D status and disease activity in other dermatoses remain variable across studies [7,18,19]. Therefore an updated synthesis integrating cutaneous mechanisms with clinical evidence across several common dermatological diseases remains warranted [4,7,8].

AIM

The aim of this paper is to present the current state of knowledge regarding the role of vitamin D in dermatology with particular emphasis on psoriasis atopic dermatitis acne vulgaris and skin cancers and to outline therapeutic perspectives based on the available clinical evidence [4,7,18,19,32,33,34].

METHODS

This article is a narrative literature review. The methodological approach was aimed at a qualitative and integrative synthesis of existing experimental and clinical evidence and did not follow the principles of a systematic review or meta analysis. No review protocol was registered and PRISMA guidelines were not applied.

Literature was identified through non systematic searches of the PubMed Scopus and Google Scholar databases. The primary focus was on publications published between 2012 and 2025 in order to reflect contemporary concepts of vitamin D metabolism and its role in dermatology. Earlier landmark publications were additionally included when required to describe fundamental biological mechanisms of vitamin D synthesis metabolism receptor signaling and action in the skin. The review was restricted to publications available in the English language.

The included literature comprised experimental in vitro and in vivo studies clinical observational studies randomized and non randomized clinical trials narrative reviews and meta analyses that addressed at least one of the following topics: cutaneous synthesis and metabolism of vitamin D expression and function of the vitamin D receptor in epidermal and immune cells mechanisms of epidermal differentiation immune modulation or antimicrobial defense related to vitamin D or clinical data describing associations between vitamin D status supplementation or topical vitamin D analogues and dermatological diseases. Selection of publications was based on the authors' expert assessment of relevance to the topic and clarity of the reported methodology and results.

The thematic scope of the review was limited to dermatological conditions most frequently discussed in relation to vitamin D and cutaneous biology, specifically psoriasis atopic dermatitis acne vulgaris and skin cancer.

Excluded publications comprised studies unrelated to skin physiology or dermatological disease articles focusing exclusively on musculoskeletal or general endocrine outcomes without relevance to the skin experimental studies without clear dermatological context editorials opinion pieces narrative commentaries conference abstracts and publications lacking sufficient methodological description to allow meaningful interpretation. Studies addressing dermatological conditions outside the defined scope were also excluded.

The review was based exclusively on published data. No primary data were collected and no reanalysis of original datasets was performed. No predefined search strings no formal inclusion or exclusion criteria with quantitative thresholds and no standardized assessment of study quality or risk of bias were applied. The included literature was analyzed descriptively and interpreted in terms of biological plausibility consistency of findings and clinical relevance. The purpose of the review was to summarize the current state of knowledge rather than to establish causal relationships or formulate clinical recommendations.

RESULTS

SYNTHESIS OF VITAMIN D AND ITS ROLE

The epidermis is one of the most important sources of vitamin D for the human body. Under the influence of sunlight —specifically ultraviolet (UV) radiation (action spectrum 290–315/280–320 nm, or UVB)—7-dehydrocholesterol (7-DHC) is converted into previtamin D_3 through a photochemical reaction in keratinocytes of the basal and spinous layers of the epidermis. Previtamin D_3 is subsequently transformed into vitamin D_3 (cholecalciferol) via thermal isomerization [2]. Following cutaneous synthesis, vitamin D_3 enters the bloodstream primarily bound to the vitamin D–binding protein (VDBP). In contrast, after intestinal absorption, vitamin D_3 is transported in association with both VDBP and lipoproteins [2,5]. Regardless of whether it is synthesized in the skin or obtained from dietary sources, vitamin D_3 is biologically inactive and undergoes two successive hydroxylation reactions to acquire full hormonal activity. First, in the liver, the enzyme vitamin D 25-hydroxylase (CYP2R1) converts it into 25-hydroxyvitamin D (25(OH)D), also known as calcidiol. Subsequently, in the kidneys, the enzyme 1α-hydroxylase (CYP27B1) converts it into the active metabolite 1,25-dihydroxyvitamin D (1,25$(OH)_2$D), also referred to as calcitriol. Both 25(OH)D and 1,25$(OH)_2$D may undergo metabolic deactivation through hydroxylation by the enzyme 24-hydroxylase (CYP24A1) [4]. Vitamin D status is assessed by measuring serum 25(OH)D levels, which represent the predominant circulating metabolite of vitamin D [6,7].

VITAMIN D AND THE EPIDERMIS

The key cells of the epidermis are keratinocytes, which, in addition to producing vitamin D, contain the enzymes CYP27A1 (25-hydroxylase) and CYP27B1 (1α-hydroxylase), responsible for converting vitamin D_3 into its active form, 1,25$(OH)_2$D. They are the only cells in the body capable of synthesizing vitamin D_3 from its precursor, 7-DHC, and converting vitamin D_3 into the active metabolite 1,25$(OH)_2$D [7]. Keratinocytes, like many other cell types, express the vitamin D receptor (VDR), which enables them to respond to locally produced 1,25$(OH)_2$D through both autocrine and paracrine mechanisms [7]. VDR is most active in keratinocytes during cellular differentiation and proliferation, regulating epidermal proliferation in the basal layer and promoting the gradual differentiation of keratinocytes as the upper epidermal layers are formed [4,7,38]. Through VDR, 1,25$(OH)_2$D regulates every stage of the differentiation process. In vivo and in vitro studies have shown that vitamin D influences keratinocyte proliferation and differentiation in a dose-dependent manner. Interestingly, low concentrations ($\leq 10^{-9}$ M) of vitamin D promote keratinocyte proliferation in vitro, whereas higher concentrations ($>10^{-8}$ M) inhibit proliferation and promote differentiation [7]. 1,25$(OH)_2$D promotes the production of keratin 1, involucrin, and transglutaminase in the spinous layer, helping maintain proper barrier function [8]. In addition, it induces the synthesis of filaggrin, loricrin, antimicrobial peptides, and long-chain fatty acids, as well as the formation of the cornified envelope in the granular layer [4,7,8]. The process of epidermal differentiation and its regulation by 1,25$(OH)_2$D and VDR proceeds sequentially, with different genes and pathways activated in keratinocytes at various stages of maturation. Beyond its role in epidermal differentiation, 1,25$(OH)_2$D enhances the innate immune function of keratinocytes by stimulating the expression of Toll-like receptor 2 (TLR2) and its coreceptor CD14. This activation initiates a feedback loop in which TLR2 and CD14 induce the expression of CYP27B1, leading to increased production of 1,25$(OH)_2$D. In turn, this promotes the synthesis of cathelicidin, a potent antimicrobial peptide [7]. The metabolic pathways of vitamin D in the skin constitute a fundamental basis for understanding its significant impact on human cutaneous physiology.

SOURCES OF VITAMIN D

Sunlight is the natural and most important source of vitamin D for the human body. Under the influence of UVB radiation, 7-dehydrocholesterol in the skin is automatically converted into previtamin D_3, which then undergoes further transformation into vitamin D_3 [9]. The skin's ability to produce this vitamin is affected by numerous factors, including age, skin pigmentation, use of sunscreen, geographic latitude, season, and time of day [9,10]. Dietary sources rich in vitamin D_3 include animal-derived products such as fatty fish (salmon, mackerel, sardines), cod liver

oil, egg yolks, and fortified dairy products (accounting for approximately 20% of intake). Individuals at increased risk of deficiency include those with limited sun exposure, elderly individuals, and persons with health conditions affecting vitamin D metabolism. In such cases, vitamin D supplementation is often necessary [9,11,41].

VITAMIN D AND PSORIASIS

Psoriasis is a chronic autoimmune and genetic disease characterized by excessive proliferation and impaired differentiation of keratinocytes, as well as inflammation of the epidermis and dermis. It affects approximately 2–3% of the population and significantly reduces patients' quality of life. The most common form is chronic plaque psoriasis (90% of cases), presenting as erythematous, pruritic plaques covered with silvery scales. Less common variants include guttate, pustular, inverse, and erythrodermic psoriasis. Psoriatic lesions typically occur on the scalp, elbows, knees, hands, feet, nails, and genital area, and are frequently accompanied by itching and nail abnormalities such as thickening or pitting [12]. Subclinical inflammation may persist even after complete clinical clearance of lesions and can contribute to disease relapse [13,14,35]. In most cases, psoriasis is mild and lesions are localized, making topical therapy the treatment of choice. In more severe cases, topical agents serve as an adjunct to systemic therapy [13]. Although the pathogenesis of psoriasis is not yet fully understood, research indicates that it is associated with impaired function of immune cells in the skin, particularly T lymphocytes [9]. Vitamin D helps reduce inflammation by lowering the activity of Th1 lymphocytes through inhibition of IL-2 and IFN-γ production and by promoting the differentiation of Th2 lymphocytes. This shift supports the release of anti-inflammatory cytokines such as IL-4 and IL-10, thereby decreasing the overall immune response [15,35]. Vitamin D also regulates keratinocyte proliferation and differentiation via activation of VDR, which promotes the production of key barrier proteins such as loricrin and filaggrin [16,17]. This supports skin integrity and helps slow the accelerated cellular turnover characteristic of psoriatic plaques.

Calcipotriol and tacalcitol are synthetic analogues of calcitriol used in psoriasis treatment. Their efficacy stems from their pro-differentiating and antiproliferative effects on keratinocytes, in addition to their anti-inflammatory properties. Owing to their effectiveness and favorable safety profile, these analogues are widely used as first-line therapy for mild to moderate psoriasis [18].

Several studies have demonstrated an association between low serum 25-hydroxyvitamin D (25(OH)D) levels and the occurrence of psoriasis [7,43]. Chandrashekar et al. and Maleki et al. reported that individuals with psoriasis had significantly reduced 25(OH)D concentrations compared with healthy controls. A negative correlation with PASI scores was also observed, suggesting that lower vitamin D levels are associated with more severe disease manifestations [19]. However, not all studies agree. Some investigations found no significant differences in vitamin D levels between psoriatic patients and healthy individuals. Based on currently available evidence, it remains unclear whether vitamin D deficiency contributes to the development of psoriasis or is a consequence of the disease itself [7].

In summary, high-dose oral vitamin D supplementation may have therapeutic value, for example in preventing loss of bone mineral density and managing psoriasis-associated comorbidities. Nevertheless, topical vitamin D preparations used alone or in combination with topical corticosteroids- particularly betamethasone [20,42] -remain the safer and preferred treatment option. Novel analogues that specifically target cutaneous vitamin D signaling pathways represent a promising direction for future psoriasis therapies.

VITAMIN D AND ATOPIC DERMATITIS (AD)

Atopic dermatitis (AD) is a common chronic and recurrent inflammatory dermatosis. It most often manifests in infancy or early childhood and may show partial or complete remission over time. Approximately 10% of affected individuals continue to experience symptoms into adulthood, where the disease may present with a more severe phenotype, significantly impairing quality of life. AD is characterized by recurrent lesions, eczematous eruptions, and pruritus. In infants, symptoms most commonly involve the face and cheeks, while in adults they typically affect the eyelids, hands, and flexural areas [21,22,23]. The pathogenesis of AD involves defects in the epidermal barrier and immune dysregulation, along with alterations in the skin microbiome [24,40]. In AD, the immune system displays increased expression of type 2 cytokines such as IL-4, IL-5, and IL-13, which have a significant impact on skin barrier function. A major epidermal protein, filaggrin, is essential for the structure and function of the stratum corneum—the outermost epidermal layer. Studies indicate that filaggrin is a key factor in AD pathogenesis, as mutations in the filaggrin gene are associated with more severe disease [26,35,37]. Barrier dysfunction results in greater susceptibility to colonization by microorganisms such as *S. aureus* and *S. epidermidis*[23,25].

Vitamin D plays an important role in both the risk of developing AD and the severity of the disease, owing to its involvement in skin physiology and immune regulation. It contributes to the formation of the stratum corneum and supports epidermal barrier integrity through lipid synthesis. Vitamin D also participates in the production of the antimicrobial peptide cathelicidin, levels of which are reduced in AD [27].

An epidemiological correlation has been observed between the prevalence of AD and higher latitudes, where reduced sunlight exposure leads to decreased endogenous vitamin D production [9]. Some studies have reported that low

serum vitamin D levels—particularly during winter months—are associated with disease progression and increased severity [28]. It is important to note, however, that AD is a multifactorial disease, and its pathogenesis involves numerous contributors beyond vitamin D, including environmental and genetic factors.

VITAMIN D AND ACNE VULGARIS

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous unit with a recurrent course. It is estimated to affect 9.38% of the global population, with approximately 85% of cases occurring during adolescence [7]. Acne primarily manifests in areas rich in sebaceous glands—such as the face, neck, chest, upper back, and shoulders. Lesions may appear as inflammatory changes, including papules, pustules, and nodules, or as non-inflammatory lesions such as open and closed comedones. Among the key factors involved in acne pathogenesis are follicular hyperkeratinization, increased sebum production and altered sebum composition, and colonization by *Cutibacterium acnes*. Studies have shown that underlying these processes are primary inflammatory and immunological mechanisms. Acne pathogenesis involves the release of pro-inflammatory mediators, including interleukins IL-1, IL-6, IL-8, IL-10, IL-12, and TNF-α. Interaction among these factors promotes the formation of the microcomedone, the precursor of all acne lesions [7,29,30,35]. External factors—such as diet, occupational exposures, psychological stress, lifestyle, cosmetics, medications, pollution, and climatic conditions—should also be considered [31].

Vitamin D plays a significant role in the pathogenesis of acne vulgaris. Sebocytes, the cells responsible for sebum production, express receptors capable of binding the active form of vitamin D, calcitriol. This interaction reduces the secretion of pro-inflammatory mediators involved in acne lesion development. A correlation has also been observed between pharmacologically elevated concentrations of $1,25(OH)_2D$ and inhibition of sebocyte proliferation [32,36]. Taken together, these findings suggest that vitamin D supplementation may effectively reduce acne lesions by modulating inflammation and suppressing sebaceous gland hyperplasia. Vitamin D additionally decreases excessive cellular proliferation by inhibiting the release of pro-inflammatory cytokines. It also regulates keratinocyte apoptosis—keratinocytes similarly express receptors for the active form of vitamin D—thereby potentially reducing the number of cells that contribute to the formation of initial acne lesions [7,36].

VITAMIN D AND SKIN CANCER

It is well established that UV radiation is the most significant carcinogenic factor for the skin, particularly in the development of melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC). At the same time, UV radiation is essential for the synthesis of vitamin D in the human body [33,44]. As previously noted, calcitriol participates in the differentiation of epidermal keratinocytes, which may help prevent tumor development [34]. However, studies have shown that malignant tumor cells, including squamous cell carcinoma (SCC) cells, may exhibit resistance to calcitriol despite normal expression of VDR receptors [34]. Overall, there is currently no substantial evidence supporting a protective role of vitamin D in the prevention of skin cancer. Nevertheless, it remains important to consider both the beneficial and harmful aspects of UV radiation and its effects on human skin.

For a clear summary of the key role of vitamin D in the dermatological diseases discussed in the Results section, a summary table with the corresponding literature sources is presented below.

Table 1. Vitamin D and dermatological diseases with reference numbers

Disease	Mechanisms described in the literature	Type of studies represented in REFERENCES	Reference numbers
Psoriasis	Keratinocyte proliferation and differentiation via VDR. Immunomodulation of T cell mediated inflammation. Use of topical vitamin D analogues. Associations between serum 25(OH)D and disease severity.	Narrative reviews. Experimental studies. Observational clinical studies. Randomized and non randomized clinical studies.	1, 4, 7, 12, 13, 14, 16, 18, 19, 42, 43
Atopic dermatitis	Epidermal barrier function. Filaggrin related mechanisms. Antimicrobial peptide induction. Immune modulation and microbiome interactions.	Narrative reviews. Experimental studies. Observational studies. Systematic reviews and meta analyses.	4, 7, 15, 21, 22, 23, 24, 25, 26, 27, 28, 40

Acne vulgaris	Sebocyte biology. Inflammatory pathways in pilosebaceous units. Effects of vitamin D on sebocyte proliferation and cytokine production.	Experimental studies. Narrative reviews. Observational studies.	1, 29, 30, 31, 32, 35
Skin cancer	Vitamin D signaling in keratinocyte differentiation. Interaction with UV exposure and carcinogenesis. Prognostic associations in melanoma.	Experimental studies. Narrative reviews. Systematic reviews and meta analyses.	3, 8, 33, 34, 44

DISCUSSION

This narrative review summarizes current knowledge on the role of vitamin D and its active metabolite $1,25(OH)_2D$ in skin physiology and in selected dermatological diseases, with an emphasis on integrating molecular mechanisms and available clinical observations rather than generating evidence based recommendations. The reviewed literature consistently demonstrates that the skin is not only a site of vitamin D synthesis but also an active target organ in which vitamin D signaling participates in regulation of keratinocyte proliferation and differentiation, immune responses, and innate antimicrobial defense.

Experimental data provide a coherent biological framework for these effects. Keratinocytes possess the enzymatic machinery required for local activation of vitamin D and express the vitamin D receptor, enabling autocrine and paracrine signaling within the epidermis. These mechanisms plausibly explain the antiproliferative, pro differentiating, and immunomodulatory effects observed in vitro and in animal models, as well as the established clinical efficacy of topical vitamin D analogues in hyperproliferative inflammatory dermatoses [2,5,15,18].

Among the conditions discussed, psoriasis represents the area with the most robust and clinically actionable evidence. The effectiveness of topical vitamin D analogues in reducing epidermal hyperproliferation and inflammation is well documented and supported by long standing clinical use. In contrast, studies evaluating serum 25(OH)D levels in patients with psoriasis show inconsistent results. While several observational studies report an association between lower vitamin D levels and greater disease severity, others fail to confirm this relationship. These discrepancies suggest that vitamin D deficiency is unlikely to represent a primary causal factor in psoriasis and may instead reflect comorbidities, lifestyle related factors, or disease associated behavioral changes such as reduced sun exposure[7,18,20,42]

In atopic dermatitis, available evidence suggests that vitamin D may influence disease expression through effects on epidermal barrier integrity and innate immune responses, including regulation of filaggrin expression, lipid synthesis, and antimicrobial peptide production. Epidemiological observations linking higher disease prevalence or severity to reduced UVB exposure and seasonal variation in vitamin D levels support a potential contributory role. However, atopic dermatitis is a highly heterogeneous and multifactorial condition, and the contribution of vitamin D signaling cannot be separated from genetic susceptibility, environmental exposures, and immune dysregulation. Clinical studies of supplementation report variable and sometimes modest effects, which limits the strength of therapeutic conclusions [9,27,28,37].

For acne vulgaris, mechanistic studies indicate that vitamin D may modulate sebocyte proliferation and inflammatory signaling pathways, providing a plausible biological rationale for a potential adjunctive role. Nevertheless, clinical evidence remains limited and largely indirect, and current data do not support routine therapeutic use of vitamin D for acne beyond correction of established deficiency [7,29,30,32,36].

The relationship between vitamin D and skin cancer remains complex and unresolved. Although vitamin D signaling participates in pathways regulating cellular differentiation, ultraviolet radiation remains the dominant and well established carcinogenic factor for the skin. Available evidence does not support a protective effect of vitamin D against the development of cutaneous malignancies, and experimental antitumor effects of calcitriol appear to be context dependent and limited by resistance mechanisms in tumor cells. Consequently, intentional ultraviolet exposure for the purpose of increasing vitamin D levels cannot be justified from a dermatological or oncological standpoint [33,34].

LIMITATIONS

This review has several important limitations. As a narrative review, it does not follow a systematic search strategy and does not include formal assessment of study quality or risk of bias. The analyzed literature is heterogeneous with respect to study design populations vitamin D assessment methods and clinical endpoints, which precludes causal inference and quantitative comparison of results. In addition, much of the evidence outside psoriasis is derived from

observational or experimental studies, limiting its direct clinical applicability. These limitations should be considered when interpreting the findings and underscore the need for well designed prospective clinical trials in this field.

CONCLUSIONS

This narrative review highlights the complex and context dependent role of vitamin D in skin physiology and dermatological diseases. Experimental evidence consistently supports the involvement of vitamin D signaling in epidermal differentiation immune regulation and antimicrobial defense, confirming the skin as both a site of vitamin D metabolism and a functional target organ.

From a clinical perspective, the most robust and reproducible evidence relates to psoriasis, where topical vitamin D analogues represent an established and effective therapeutic option supported by long term clinical use. In contrast, associations between serum 25(OH)D levels and the course of other inflammatory dermatoses, including atopic dermatitis and acne vulgaris, are heterogeneous and largely derived from observational or mechanistic studies. These data do not allow causal conclusions or uniform therapeutic recommendations.

Vitamin D deficiency is more likely to act as a modifying or accompanying factor in chronic skin diseases rather than as a primary pathogenetic driver. Consequently, routine vitamin D supplementation for dermatological indications alone cannot be justified on the basis of current evidence. Assessment of serum 25(OH)D levels and correction of confirmed deficiency may be considered in selected patients as part of general medical care, but should not replace disease specific standard therapies.

Overall, the available evidence underscores the need for cautious interpretation of experimental and epidemiological findings and reveals significant gaps in high quality interventional data. Future research should focus on well designed prospective and randomized clinical studies with clearly defined dermatological endpoints, standardized assessment of vitamin D status, and careful differentiation between systemic supplementation effects and local cutaneous vitamin D signaling. Such studies are necessary to clarify the clinical relevance of vitamin D in dermatology beyond its established role in psoriasis.

DISCLOSURE

AUTHORS' CONTRIBUTIONS

All authors have made substantial intellectual contributions to the work and approved it for publication.

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USE OF ARTIFICIAL INTELLIGENCE

The authors declare that no artificial intelligence tools were used in the generation, writing, editing or revision of this manuscript. All content was created solely by the authors.

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CONFLICT OF INTEREST

Authors declare no conflicts of interest.

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