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# Application of Vitamin E in Skin Nourishment: A Review

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**ABSTRACT**

In dermatology, vitamin E is a significant fat-soluble antioxidant that has been used for more than 50 years. It is a crucial component of numerous cosmetic items. Through its role as a free-radical scavenger, it shields the skin against a variety of harmful effects caused by sun radiation. Research from experiments indicates that vitamin E has both photoprotective and antitumorigenic qualities. Few controlled clinical trials exist that offer a justification for using vitamin E in dermatology practice with clearly established dosages and clinical indications. This article's goal is to explore vitamin E's clinical and cosmetic consequences in dermatology.

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## 1. INTRODUCTION:

Most people are aware of vitamin E as the most well-known fat-soluble non-enzymatic antioxidant because of its capacity to prevent pro-oxidant agents produced by reactive oxygen species (ROS) from being active. Free radicals produced by can be eliminated with vitamin E. avoiding the harmful effects of endogenous and/or exogenous substances, such as medications, pollutants, and UV radiation. Vitamin E's antioxidant properties are closely related to its capacity to prevent unsaturated fatty acids' lipid peroxidation. acids, which integrate with cell membranes and so block lipid oxide production [1,2]. Alpha-tocopherol, a form of vitamin E, possesses antioxidant properties that are attributable to due to its capacity to primarily react with the radical peroxyl (HOH•) as well as singlet oxygen ($^1O_2$) that promotes the peroxidation of lipids. The process of scavenging free radicals takes place.by means of the establishment of a steady.

Tocopheroxyl is a low-energy radical that cannot react with the substance that forms free radicals [3]. Tocopherol Acetate is the primary agent that may. But the transformation into the pure (isolated) For the skin to have the intended effects, shape eliminate peroxyl radicals from lipid membranes, including LDL (low-density lipoprotein) membranes [4]. Alphatocopherol, in its refined form, or its derivatives, is a traditional component in dermatologyis necessary. Applications that are topical are created to treat melasma, shield the skin from ultraviolet radiation (UVR), and reducing the effects of ageing [5, 6]; the effects on skin are amplified when vitamin E is combined with additional antioxidants [7]. A low vitamin E diet may be linked to skin diseases, according to certain research. For several skin conditions, including cutaneous ulcers, epidermolysis bullosa, yellow nail syndrome, pressure ulcers, burns, scleroderma, morphea, calcinosis cutis, Raynaud's phenomenon, and inflammatory disorders. The oral vitamin E supplementation may lessen melasma pigmentation and contact dermatitis lesions also showed signs of atopic dermatitis remission, prevention of the sunburn reaction and the ensuing long-term skin damage [5]. When vitamin E is paired with other antioxidants, it can help with photoprotection and slow down the progression of melanoma by encouraging tumour cell death and preventing VEGF-mediated angiogenesis. Additional outcomes with alpha-tocopherol: enhancement of brightness, roughness, and periorbital fine lines clinical measures of skin tone, elasticity, density, collagen production, and overall appearance assessments of the skin. Tocopherol acetate used topically dramatically lowers the degree of skin irritation, erythema, and edoema brought on by sunburn by UVB [8]. Determining the antioxidative action of vitamin E in vivo poses a

challenge. since the skin naturally contains it, but further research used the isolated form and its metabolites can be investigated in topical medications [9].

**2. Role of vitamin E in skin's Oxidative stress:**

The phenolic hydroxyl (OH) in vitamin E's structure, which can chemically mediate the peroxyl radical (PUFAOO*) and donate hydrogen to form a stable lipid species (PUFAOOH), is the mechanism of action (Figure) for vitamin E's antioxidant activity in the skin. Consequently, vitamin E becomes a comparatively non-reactive free radical when it donates the hydroxyl H. Vitamin E and lipid peroxidation mechanism in cells. $O_2$ is oxygen; PUFA, polyunsaturated fatty acids; VE-OH, vitamin E, alpha-tocopherol; VE-O*, radical tocopheroxyl; OH*, oxygen radical - hydroxyl; PUFA*, lipid radical; PUFAOO*, Peroxy lipid radical.

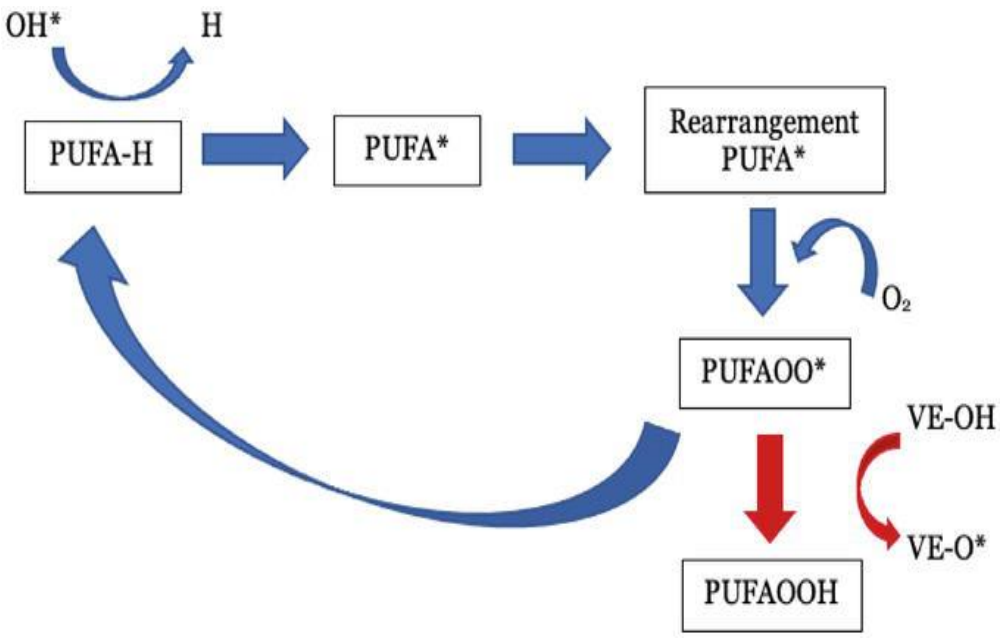



**Fig. Vitamin E and lipid peroxidation mechanism in cells. O2 is oxygen; PUFA, polyunsaturated fatty acids; VE-OH, vitamin E, alpha-tocopherol; VE-O*, radical tocopheroxyl; OH*, oxygen radical - hydroxyl; PUFA*, lipid radical; PUFAOO*, Peroxy lipid radical**.

When the single electron travels to the ring of aromatic compounds as a result, integration happens in biological membranes by electronic displacement and is incredibly close to the phospholipids in cell membranes that contain polyunsaturated fatty acids, which stops the chain reaction. By donating hydrogen from the OH group to the unsaturated lipid or the lipid

peroxyl radical (PUFOO *), vitamin E is able to halt the reaction and create the low-energy tocopheroxyl stable radical (VE-O *), which cannot function as a free radical forming agent [40, 41]. By directly interacting with the singlet oxygen and superoxide anion of other lipid radicals, vitamin E also exhibits antioxidant activity against them [19].Research has demonstrated the role of vitamin E in regulating the damage caused by FR caused by UVR's effect on the skin, including photoaging, lipid peroxidation, immunosuppression, and photocarcinogenesis [42]. By inhibiting the synthesis of prostaglandin, pro-inflammatory cytokines, cyclooxygenase-2 (COX-2) and NADPH oxidase, vitamin E can lessen the inflammatory responses of the skin [43–45].Apart from its anti-inflammatory properties, vitamin E can also regulate the signalling pathways of protein kinase C (PKC) and phosphatidylinositol 3-kinase (PI3-K), as well as lessen the upregulation of collagenase production. The connection between vitamin E (alpha-tocopherol) and PKC protein does not happen directly; instead, it happens preventively to its activity at the cellular level. PKC regulation may be important in terms of controlling cell development [45, 46].

Through its inhibition of metalloprotein 1 (MMP-1), which is involved in the early step of collagen hydrolysis, vitamin E has the capacity to considerably decrease collagen degradation [44]. Being one of the primary antioxidants in the human epidermis, it is thought to have an action to reduce the process of photocarcinogenesis because it is found in deeper layers of the skin. Utilising vitamin E as a very sensitive and early measure for environmentally induced oxidative damage is one of its other qualities [47,48]. As a result, vitamin E stops the breakdown of fatty acids and lipoperoxidation of cell membranes, both of which are necessary for the body and skin to function properly [8, 49, 50]. Vitamin E can stop FR caused by UVA rays, shield endogenous antioxidants from deteriorating mechanisms, and stop lipid peroxidation and lessen UVR-induced immunosuppression. Vitamins E and C have been linked to increased protection against sunburn and erythema, as well as potential protection against skin cancer and ageing of the skin. Applying vitamin E topically before going outside helps prevent the UVB-induced cyclobutane pyrimidine dimer (CPD) from forming on the skin [46]. The endogenous mechanism is insufficient to prevent harmful skin damage because excessive exposure to pollution and UV radiation generally increases the production of free radicals. This calls for the oral and/or topical supplementation of antioxidant substances, such as vitamin E.[51]

**3. Dermatology Indication:**

- **Condition of yellow nails**

IV degree of evidence: The yellow nail syndrome is characterised by lymphedema, slowly developing, opaque yellow nails with an accentuated yellow curvature, With long-term respiratory conditions such persistent bronchitis, chronic sinusitis with pleural effusions.[11] Among these is vitamin E. the yellow nail syndrome therapy options,[12] in a dosage of 1000 IU once daily for a duration of half a year.[13]Hemolysis and headache caused by dapsone: (Level of evidence (IV) Numerous investigations to determine the preventive impact of vitamin Regarding the hemolysis brought on by dapsone therapy, it was observed that 800 IU/day of (dl-α-tocopheryl acetate)provides a limited degree of protection against dapsone-induced hemolysis in individuals suffering from herpetiform dermatitis.[14,15] Vitamin E has also been applied to headaches brought on by dapson[e][16]. One known impact of methemoglobinemia is headache, which is apparently ameliorated by vitamin E through lowering previously increased methemoglobin concentration. Symptom, since increased levels of methemoglobin appear to be the laboratory parameter that is most reliable in investigations of vitamin E to guard against adverse effects of dapsone.[14]

- **Dermatoses with pustular subcornea: (Level of evidence IV)**

D-α-tocopheryl acetate, or vitamin E, 100 IU daily, progressively boosting to 400 IU per day for four weeks is among the medicinal methods in subcutaneous pustular dermatoses, specifically those who don't respond well to traditional drugs.[17]

- **Amyloidosis cutaneous: (Level of evidence IV)**

A combination of tocopherol and retinoic acid is called tocoretinate. In an investigation meant to assess the impacts of topical to gather information about macular and lichen amyloidosis, Topical tocoretinate was found to decrease the clinical signs of macular amyloidosis and lichen.[18]

- **Other dermatological conditions for which vitamin E**

This is not very helpful Atopic dermatitis, a study with a single blind and placebo control was carried out by Tsoureli-Nikita et al., wherein 96 individuals with atopic dermatitis were administered oral vitamin E (400 IE/day) or a placebo for eight months. They discovered a decline and almost complete remission of A 62% reduction in serum IgE levels in individuals

with atopic dermatitis the group receiving vitamin E. IgE serum levels in atopic individuals are lowered by vitamin E.[19] IgE levels, vitamin E consumption, and atopy's clinical symptoms are all correlated that atopic dermatitis may benefit from vitamin E treatment.

- **Disease Hailey-Hailey**

Three patients with Hailey-Hailey disease were reported to have had their conditions under control by oral medication in 1975 by Ayres and Mihan. amounts of vitamin E as d-α-tocopheryl acetate in amounts of 800–1200 IU/L [20] The precise process by which Vitamin E Although it is unclear what causes this illness, its antioxidant action in preventing lipid peroxidation from damaging the cell membrane, so maybe halting the production of autoimmune antibodies could potentially be a crucial element.[21]

- **Bulosa epidermolysis**

Numerous case studies point to the effectiveness of vitamin E (300–600 IU daily) in the treatment of epidermolysis bullosa.[22, 23] Vitamin E functions as an antioxidant, shielding the membranes of cells and organelles found inside cells due to lipid peroxidation.[24] It is conceivable. that epidermolysis bullosa is caused by a genetic abnormality that affects the ability of the tissues to store vitamin E of tissues to utilise it, requiring an extra supply. [24]

- **Psoriasis**

A natural remedy for psoriasis called "Mirak" has lately been accessible in various European nations. Valconic soil, pure spring water, and vitamin E cream make up Mirak. It has no major side effects and produces a moderate therapeutic benefit when compared to a placebo, but it might not be able to compete with the psoriasis treatments that are now available.[25]ulcers on the skinFour patients have shown improvement with vitamin E treatment at doses of 800 IU/L, progressively increasing to 1600 IU/L.[26]

- **Preventing skin cancer**

Research on mice revealed that when mice were fed α-tocopherol acetate, UV-induced tumours were inhibited.[27] Numerous investigations on humans have not revealed any impact of vitamin E on the development or prevention of skin cancer.[28, 29]

- **Wound recovery**

Oral therapy for burns and pressure ulcers include vitamin E, zinc, and vitamin C.[30] It has been shown that antioxidant supplementation with vitamins E and C as well as the mineral zinc appears to improve antioxidant defence against oxidative stress and shorten the healing period after injury.[31]

- **Melancholy**

Vitamin E by itself hasn't proven to be very effective in treating melasma.[32] Depigmentation has been demonstrated to be caused by interfering with the melanocyte membranes' lipid peroxidation, a rise in the amount of intracellular glutathione and a reduction in tyrosinase [33] Within a double-blind, randomised, placebo-controlled study, an oral proanthocyanidin in conjunction with vitamins A, C, and E was evaluated in sixty female Filipinos who had bilateral epidermal Mascara. For eight weeks, the antioxidants were given twice a day and were contrasted using mexametric analysis with placebo ingestion. Analysis of the Melasma Area and Severity (MASI) score. [34] There was a notable decline in pigmentation and MASI scores. by mexametry in the areas of Malar.

A standardised extract of the bark of the French maritime pine (Pinus pinaster), pyrogenol is a well-known and powerful antioxidant that is several times stronger than vitamin E. It also boosts the endogenous antioxidant enzyme system and regenerates vitamin E. Consequently, a clinical trial was conducted to determine its effectiveness in treating melasma. Thirty melasma-affected women took one 25 mg tablet of pycnogenol with meals three times a day for a total of thirty days. Clinical evaluations of these patients were conducted using metrics such the pigmentary intensity index, melasma area index, and standard blood and urine testing. Following a 30-day course of treatment, patients' average melasma area shrank by 25.86 ± 20.39 mm (2) ($P < 0.001$), and there was a 0.47 ± 0.51 unit decrease in the average pigmentary intensity.[35 Derivatives of α-toc suppress melanogenesis in epidermal melanocytes and tyrosinase in vitro [36, 37].[37] The depigmenting effect of α-Toc may be explained by its antioxidant qualities, which prevent melanocyte membranes from peroxiding their lipids and raise the amount of glutathione within cells. [38]

- **Vulgaris acne**

One of the trials, which involved 98 patients, focused on correcting the sebaceous follicle keratinization with a mix of vitamins C and E.[39] This was observed to stop the development of comedones, preventing the Propionibacterium from a culture medium's acne. Lipid peroxidation is prevented by vitamin E. of serum from follicle-induced leaking caused by bacteria and sebaceous glands, avoiding inflammation brought on by irritation caused by peroxide.High doses of isotretinoin have also been used with vitamin E. to lessen the adverse effects of isotretinoin. Still, Research has indicated that vitamin E has no discernible reduce the negative effects of retinoid when taken with isotretinoin when treating acne.[40,41]

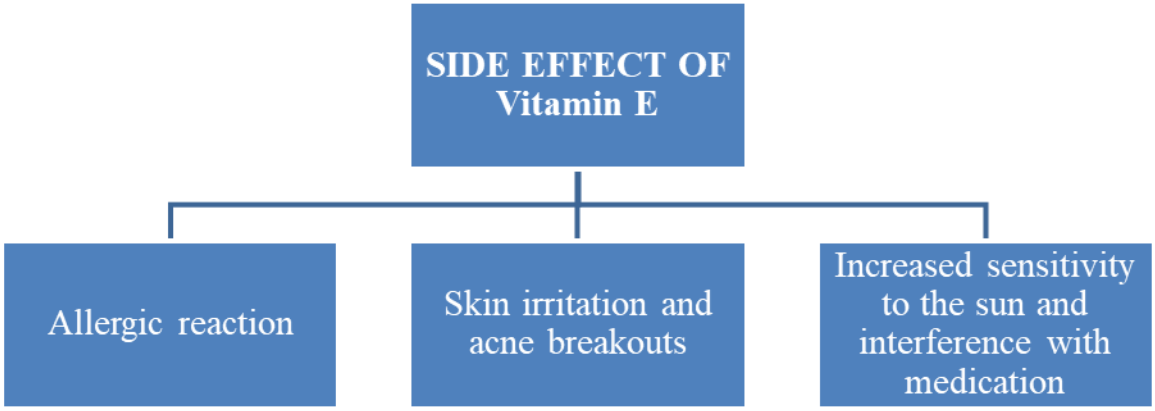
- **Sclerosis**

Scleroderma patients have a markedly higher level of oxidative stress in comparison to healthy control indicating that oxidative damage caused by free radicals happens in scleroderma.[42] Vitamin E and other antioxidants may, Consequently, be advantageous. Additionally, vitamin E is thought to stabilise lysosomal membranes, perhaps preventing action relate to the process of autoimmune disease.[21]The use of vitamin supplements has improved Despite not being dermatological, the skin of scleroderma patients The condition of scleroderma did not get better.[43] Some scleroderma constituents, such as morphea, The conditions Raynaud's phenomenon and calcinosis cutis react to vitamin E [44] In these instances, the vitamin E dosage varied. between 200 and 1200 IU daily. A 45-year-old man who had Raynaud's phenomenon, likely early scleroderma, and ulceration and gangrene of the fingertips was one of the patients who were effectively treated. He was given 800 IU. Vitamin E (50 IU per millilitre) orally every day and applied the vitamin to the ulcerated fingers twice a day. The sores grew less painful, after two weeks and recovered nearly fully in a month. [45]

**4. Side Effect:**

When taking the prescribed daily dosage, the majority of people do not suffer any negative effects. An excessive dosage may result in stomach pains, nausea, diarrhoea, exhaustion, and weakness headache, rash, bruises, bleeding, and blurred vision. Since vitamin E is fat-soluble, administering an amount over the body's daily requirement causes buildup inside the body, which leads to hypervitaminosis E. For more than a year, healthy persons who take 100 mg of vitamin E daily run the risk of developing hypervitaminosis E, which is characterised by

decreased platelet aggregation and disruption of vitamin K metabolism, which can lead to bleeding tendencies.[60] Rarely, topical vitamin E treatment might result in xanthomatous response, erythema multiforme, and contact dermatitis. [61, 62].[63]




## 5. Conclusion:

Although novel formulations for use in skin care and cosmetics have been developed, controlled clinical trials supporting well-defined doses and therapeutic indications for oral and topical vitamin E are scarce. It remains unclear whether the millions of dollars' worth of vitamin E products that customers and patients have paid for have been beneficial after so many years of research on the nutrient. Improved knowledge of this vitamin could be useful in assessing the recommended uses and dosage schedules for the management of both acute and long-term skin conditions.

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