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REVIEW ARTICLE

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Vitamin Deficiencies and Their Impact on Skin Function

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Abstract

Vitamins are essential dietary items because of their functions as enzyme cofactors and catalysts in the body. Vitamin deficiency can significantly impact the skin, leading to a variety of dermatological problems, such as dryness, premature aging, hyperpigmentation, palmoplantar keratoderma, and even nail disorders. Alcoholism, rigid diets, inadequate parental nourishment, and intestinal absorption issues may all be factors in vitamin deficits. The most commonly deficient vitamins in relation to skin health are vitamin A, biotin, vitamin C, and vitamin D. Vitamin A helps to maintain the skin's barrier function and promote skin cell growth, while biotin helps to support the skin's underlying structure. Vitamin C is essential for collagen synthesis and skin brightening, and vitamin D is a crucial nutrient for overall health, including the skin. A balanced and varied diet and proper skin care can help prevent and address vitamin deficiencies, keeping the skin healthy and radiant. This review examines the most common vitamin deficiencies and their impact on the skin.(**International Journal of Biomedicine. 2023;13(2):205-209.**)

Keywords: skin • malnutrition • vitamin deficiency • dermatological signs

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Introduction

Vitamin deficiency can significantly impact the health and appearance of the skin. Vitamins are crucial in maintaining skin health, as they support various cellular processes that keep skin youthful, radiant, and healthy.[1] Some of the most common skin-related issues associated with vitamin deficiencies include wrinkles, dullness, uneven skin tone, dark circles, and even skin diseases like psoriasis and eczema.[2] It is important to understand the role vitamins play in skin health so that any potential deficiencies in the diet can be identified and addressed. By consuming a balanced diet rich in vitamins and antioxidants, people can help maintain healthy skin and prevent the negative impacts of vitamin deficiency on the skin. This study demonstrates the prevalence of vitamin deficiency and its dermatological consequences.

Fat-Soluble Vitamins

Vitamin D

Vitamin D is known to be essential for bone health as well as being a hormone that regulates a number of physiological processes. It can be produced by food sources or, when ultraviolet (UV) light is present, via the skin's cholesterol progenitors. This hormonally inactive version needs two further hydroxylation processes to become active. The initial conversion to 25-hydroxyvitamin D, the most active and abundant version of vitamin D within the body, happens in the liver and numerous other organs.[3] Vitamin D's physiologically active, hormonal form, 1,25-dihydroxyvitamin D, is produced by the kidneys and is necessary for bone health, calcium and phosphorus metabolism, and other bodily functions. Many cell types, beyond the skeletal structure, have vitamin D sensors and can transform blood 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D for regional uses, such as immunological control and cell proliferation and differentiation. Vitamin D nutritional status may be determined by measuring blood levels of 25-hydroxyvitamin D. The ideal levels are in the 75–125 nmol/L range.[4]

Deficiency

According to the United States Endocrinal Association, a vitamin D level of 21 to 29 ng/mL is inadequate, and a level of 20 ng/mL is deficient. Vitamin D status is influenced by UV radiation exposure, skin color, sex, body mass index, physical activity, vitamin D receptor mutations, and alcohol consumption.[3,4] Vitamin D is a crucial nutrient for overall health, including the skin. A deficiency of vitamin D may result in various skin problems and affect the appearance and function of the skin. Here are some ways in which vitamin D deficiency can impact the skin:

1. Atopic dermatitis has been connected to vitamin D receptor polymorphisms and low vitamin D levels. People with atopic dermatitis were found to have a higher incidence of vitamin D receptor gene variations than do healthy individuals, highlighting the importance of vitamin D in the development of the condition.[5] A recent study revealed that low levels of vitamin D in the blood were correlated with more severe eczema symptoms.[1]

2. Psoriasis: IFN-c, IL-2, IL-6, and IL-8 are among the inflammatory mediators that are inhibited by 1,25-dihydroxyvitamin D, while IL-10, an anti-inflammatory cytokine, is increased. These actions help to limit the inflammatory process in the pathogenesis of psoriasis.[3] Furthermore, vitamin D's immune regulatory ability aids in modulating and controlling the levels of antimicrobial peptides involved in the pathogenesis of psoriasis. A recently published study demonstrated that inadequate vitamin D levels had been linked to an increased chance of developing psoriasis and worsening symptoms in people with the condition.[6]

3. Vitiligo: At present, the pathogenic mechanism of vitiligo is not completely clear. There are studies suggesting that vitamin D may increase the melanogenesis and tyrosinase content of human melanocytes through its anti-apoptotic effect, thus preventing the loss of skin pigment.[7] A meta-analysis conducted by Zhang et al.[8] in 2018 indicated that patients with vitiligo had a lower level of 25(OH)D compared with controls. A meta-analysis performed by Varikasuvu et al.[9] showed significantly decreased Vitamin D levels in vitiligo, and its association with indoor/outdoor type of work of vitiligo patients. However, a meta-analysis performed by Song et al.[10] provided no convincing evidence for a causal effect of 25(OH)D or 25(OH)D3 levels on the risk of vitiligo. According to recent observational studies that have produced conflicting findings, it is unclear if insufficient vitamin D contributes to the development of vitiligo.[8]

4. Connective tissue disorders are also associated with a vitamin D deficit. With rates reaching 90%, vitamin D insufficiency is particularly frequent in those with systemic sclerosis. Lower vitamin D levels have also been linked to cardiovascular problems and lupus nephritis in people with systemic lupus erythematosus. Also, there is some association between Sjogren syndrome and decreased vitamin D blood levels.[11]

5. Alopecia areata: In keeping with earlier research demonstrating that vitamin D deficiency may be related to autoimmune illnesses, it has been proposed that vitamin D's immune modulatory activity contributes to alopecia areata. Most observational studies have found a significant correlation between vitamin D deficiency and autoimmune reactions in alopecia areata, with disease severity rising.[12]

6. Autoimmune bullous diseases: Vitamin D insufficiency has also been connected to autoimmune bullous diseases. Insufficiency has been shown to be more prevalent in pemphigus and pemphigoid patients, although this connection cannot be related to the onset of autoimmune bullous diseases.[13]

7. Acne: According to research, vitamin D may play a part in the development of acne. Based on one study, low vitamin D levels were linked to more severe acne.[14]

8. Wrinkles: Vitamin D is important for skin health and skin aging. According to one study, insufficient vitamin D levels were associated with increased skin aging and wrinkles.[15]

9. Pigmentation Disorders: Vitamin D is also involved in regulating skin pigmentation. One published study suggested that low vitamin D levels relate to an increased incidence of pigmentation disorders such as melasma.[16]

10. Wound healing: Vitamin D is essential for wound healing because it promotes the production of antimicrobial peptides and regulates inflammation. Low levels of vitamin D have been linked to impaired wound healing.[17]

11. Skin infections: Vitamin D has antimicrobial properties and helps the immune system respond to skin infections. Low vitamin D levels have been linked to an increased risk of skin infections.[18]

12. Skin neoplasms: Vitamin D levels have been studied in numerous tumor subtypes, and most of these neoplastic disorders have a substantial correlation with vitamin D concentrations. Recent research has revealed that cutaneous T-cell lymphoma cells express the vitamin D receptor and can undergo apoptosis in response to a significant blood vitamin D level.[19] Numerous studies have indicated a link between melanoma and vitamin D levels. Melanoma has been linked to both the receptor's polymorphisms and expression. Vitamin D prevents tumor angiogenesis, invasion, and metastasis. Patients with stage IV melanoma were found to have lower serum vitamin D levels than those with stage I melanoma. Additionally, those with insufficient vitamin D levels have higher risks of melanoma recurrence.[20] Vitamin D insufficiency has also been linked to basal cell cancer. By inhibiting Hedgehog signaling, vitamin D produced by UV radiation protects the skin from the carcinogenic effects of UV radiation. However, such a response was not observed with vitamin D taken orally. Mutations in the vitamin D receptors are also related to the development of basal cell cancer. Keeping vitamin D values over 25 ng/mL could greatly prevent basal cell carcinoma recurrence.[21]

Vitamin A

Vitamin A, or retinoid, is necessary for healthy skin and barrier functions, as well as for a proper immune system, growth and development, eyesight, and reproduction. The human diet contains two sources of vitamin A: preformed vitamin A (retinol and retinyl esters) and provitamin A carotenoids.[22,23] Retinol is a type of vitamin A obtained from animals that serves as a precursor to the body's most active form (retinoic acid). The plant forms of vitamin A, alpha-carotene, beta-carotene, and beta-cryptoxanthin, can all be transformed into retinol in the body. The liver stores vitamin A, and the total body quantity is 300 to 700g/day for children and 700 to 900 g/day for adults.[24]

Deficiency

Vitamin A is an essential nutrient for maintaining healthy skin. A deficiency of vitamin A can lead to several skin problems. Vitamin A deficiency is thought to affect around 30% of children worldwide. The most common cause of vitamin A deficiency is inadequate nutritional intake due to malnutrition. In affluent countries, people with hepatic cirrhosis, small bowel syndrome, cystic fibrosis, and

alcoholism frequently have vitamin A deficiency.[24] The risk of clinical manifestation is related to serum retinol levels of 0.35mol/L. One of the main effects of a vitamin A deficiency is xerosis, or dry skin. This is because vitamin A is necessary for sebum production, an oil produced by the skin that helps keep it hydrated. Without enough vitamin A, the skin can become dry, flaky, and itchy.[25] Another impact of vitamin A deficiency on the skin is a decreased ability to fight infections. Vitamin A is required for the immune system, and a deficiency can lead to an increased risk of infections, such as boils, impetigo, and folliculitis. In addition, vitamin A is essential for cell development and repair, and a deficiency can lead to a reduction in the rate of cell turnover. This can result in a buildup of dead skin cells, leading to a dull, rough, and uneven skin texture. Finally, vitamin A deficiency can also lead to a loss of skin elasticity, causing wrinkles and fine lines to form. This is because vitamin A helps to maintain the skin's collagen levels, which are important for skin elasticity and firmness. Vitamin A palmitate oil is used to treat vitamin A deficiency. The recommended dosages are 60,000 IU taken orally for two days, followed by 4,500 IU each day.[26]

Water-Soluble Vitamins

Vitamin B7

Vitamin B7, also known as biotin, is a water-soluble vitamin that plays a critical role in several metabolic processes in the body. This vitamin serves as a coenzyme for five carboxylases dependent on vitamin B7 and is involved in essential processes such as gluconeogenesis, lipogenesis, and amino acid breakdown. In addition, biotin is crucial for protein synthesis, including the production of keratin, which is necessary for the proper development of hair and nails. Biotin can be obtained from various dietary sources, including meats, eggs, salmon, sunflower seeds, and sweet potatoes.[27]

Deficiency

Biotin deficiency can be either inherited or acquired. The consumption of raw eggs is a common cause of acquired biotin insufficiency, as a protein called avidin, which is present in raw egg whites, binds strongly to biotin, preventing its use as an important cofactor. Other factors that can lead to acquired biotin deficiency include alcoholism, pregnancy, the use of certain medications such as valproic acid and isotretinoin, reduced absorption, or prolonged use of antibiotics, which can disrupt the natural gut flora.[28] Congenital or hereditary biotin deficiency, on the other hand, can occur due to a lack of enzymes such as biotinidase or holocarboxylase synthase, which are inherited as an autosomal recessive trait. The neonatal form, due to a deficiency in the holocarboxylase synthase enzyme, which manifests within the first six weeks of life, is a potentially fatal condition. Extensive dermatitis and severe alopecia are possible skin lesions. The infantile variant appears after three months of age and is caused by a deficiency of the enzyme biotinidase, which is important in carboxylase breakdown and subsequent free biotin absorption. It is possible to lose hair on the scalp, brows, and eyelids. In advanced instances, hair loss can be complete.[29] Alopecia, seborrhea, conjunctivitis, eczema, and numerous neurologic abnormalities, such as seizures, lethargy, and hypotonia, are common signs of biotin insufficiency. Biotin insufficiency is uncommon as it is available in a variety of foods and is also generated by organisms in the gut flora. One way to detect biotin deficiency is to measure the levels of biotinylated propionyl-CoA carboxylase and methylcrotonyl-CoA carboxylase in white blood cells. Additionally, the levels of biotin in urine and serum can also be measured as an indicator of biotin deficiency. Biotin plasma concentrations typically range from 400 to 1,200 ng/L. A plasma reading of less than 200 ng/L is considered deficient. For adults, the daily nutritional requirement for biotin is 5 mg. A varied diet containing enough biotin is usually sufficient.[30]

Vitamin C

Vitamin C, also called ascorbic acid, is an antioxidant and plays a crucial role in the hydroxylation of specific amino acids such as tyrosine and tryptophan. As these amino acids are integral components of collagen, vitamin C supports collagen synthesis, thereby aiding in the formation of the skin barrier and dermal collagen. It has anti-aging qualities as well since it guards the skin against oxidation. Vitamin C must be obtained from food; humans are unable to manufacture it due to a lack of the L-gluconolactone oxidase enzyme. The most significant food sources are fresh fruits and vegetables. Because vitamin C is heat-sensitive, cooking or boiling reduces its nutritional value. The entire body store of ascorbic acid is 1,500 mg, and symptoms of insufficiency appear when that amount falls below 350 mg.[31]

Deficiency

Alcoholism, anorexia, cigarette usage, poor nutrition, male gender, fever, viral infections, and antibiotic use are all risk factors for ascorbic acid deficiency. Scurvy is the most common symptom of insufficiency. It is induced by vitamin C's function in collagen production and the maintenance of the triple helix's collagen stability. Although scurvy is uncommon, it remains a condition that occurs, particularly in children who are receiving pasteurized milk.[32] Fatigue, mood swings, despair, irritability, and anorexia are the first indications of a lack of ascorbic acid for eight to twelve weeks. Specific dermatologic findings following these nonspecific symptoms may include enlargement of the gingiva with tooth loss, poor wound healing, hyperkeratosis, and ecchymosis. Hair irregularities, such as swan-neck and corkscrew hair, are common in scurvy due to a disturbance in the production of the disulfide bond. Perifollicular hemorrhages on the lower limbs, forearms, and belly are noticed in the early stages of the illness. When these features occur on the legs, they may cause woody edema, which is characterized by ecchymosis, discomfort, and restricted mobility. Koilonychia and splinter hemorrhages are two diseases of the nails that have been linked to ascorbic acid deficiency. Although the gums initially appear red, puffy, and glossy, they eventually turn black and necrotic in the later stages. Tooth loss is common, as are ocular manifestations such as retrobulbar hemorrhage into the optic nerves, which causes papilledema and atrophy.[33] Up to 80% of patients with this deficiency experience musculoskeletal symptoms such as muscle hematomas, arthralgia, and

myalgia. Risk factor assessment, clinical assessment, and lab testing all precede the diagnosis. The radiologic evidence will be pathognomonic. Low-plasma ascorbic acid (0.2 mg/dL) or leukocyte ascorbic acid (7 mg/dL) levels are much more accurate in diagnosis.[34] The typical therapy is vitamin C replenishment. Scurvy patients should take one to two grams of ascorbic acid daily for two to three days, followed by a daily intake of 500 mg for a week, and then a daily intake of 100 mg for one to three months. Treatment should continue until all clinical symptoms have resolved. Hair begins to respond after one month, whereas cutaneous signs take two weeks. A full recovery is anticipated after three months of ascorbic acid administration. When there is no shortage, daily needs for males, females, and breastfeeding mothers range from 90 mg to 120 mg for men, up to 45 mg for youngsters, and 75 mg for women.[35]

In conclusion, vitamins are essential for proper bodily functions, and their deficiency can result from inadequate intake, poor absorption, poor nutrient utilization, or increased breakdown. The development of mucocutaneous changes, such as skin lesions, can aid in identifying underlying vitamin deficiencies, although they may not be specific for all vitamin deficits. Scurvy is a disease caused by vitamin deficiency and associated with skin lesions that can be diagnostic. Other skin lesions commonly associated with vitamin deficiencies include cutaneous and mucosal pigmentation, palmoplantar keratoderma, and nail layering. Clinical observations and laboratory measurements of blood or urine vitamin levels can be used to diagnose deficiencies.

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Competing Interests

The authors declare that they have no competing interests.

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