

A Pilot Study to Examine Copper Deficit in Dermatological Disorders

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Introduction

A prevalent inflammatory skin condition that affects roughly 2-3% of people worldwide, psoriasis vulgaris is characterized by erythematous plaques with silver scales. Genetic susceptibility, dysregulated immunological responses, environmental triggers, and aberrant keratinocyte proliferation are all part of the complex pathophysiology of psoriasis. Even while our knowledge of the molecular pathways behind psoriasis has advanced, we still don't fully grasp the disease's etiology and pathophysiology. According to new research, vitamin K, a fat-soluble vitamin that is important for bone metabolism and blood clotting, may potentially be involved in the pathophysiology of psoriasis. Phylloquinone (vitamin K1) and menaquinone (vitamin K2) are two of the several forms of vitamin K, and each has unique biological properties [1].

Vitamin K has been shown in preclinical research to have anti-inflammatory properties, such as suppressing Nuclear Factor-kappa B (NF- κ B) signaling pathways and inhibiting the generation of pro-inflammatory cytokines. Furthermore, it has been demonstrated that vitamin K increases antioxidant enzyme activity and decreases oxidative stress, which may lessen the oxidative damage seen in psoriatic skin lesions. Furthermore, vascular calcification, a process linked to cardiovascular comorbidities associated with psoriasis, is inhibited by vitamin K-dependent proteins such Matrix Gla protein (MGP). Notwithstanding these molecular discoveries, little clinical information is known about the frequency of vitamin K shortage in psoriasis vulgaris patients and how it relates to the severity of the condition and clinical results [2].

Description

Erythematous plaques with silver scales are the result of dysregulated keratinocyte proliferation and aberrant immunological responses in psoriasis vulgaris, a chronic inflammatory skin condition. Although the precise cause of psoriasis is still unknown, mounting data points to a possible link between the pathophysiology of the condition and vitamin K insufficiency. A fat-soluble vitamin, vitamin K has a variety of physiological effects in addition to its traditional functions in bone metabolism and blood coagulation. Preclinical research has shown that vitamin K has anti-inflammatory qualities, such as suppressing NF- κ B signaling pathways linked to the pathophysiology of psoriasis and inhibiting the synthesis of pro-inflammatory cytokines. Additionally, it has been demonstrated that vitamin K regulates tissue calcification and oxidative stress, two mechanisms that aid in the onset and

advancement of psoriasis. Matrix Gla Protein (MGP) and other vitamin K-dependent proteins are essential in preventing arterial calcification, a prevalent aspect of cardiovascular comorbidities linked to psoriasis. Clinical data on the prevalence of vitamin K deficiency in psoriasis vulgaris patients and its effect on the severity of the disease and clinical outcomes are scarce, despite these molecular insights. A possible connection between vitamin K insufficiency and the pathophysiology of psoriasis has been suggested by several observational studies that found lower serum levels of vitamin K in psoriasis patients than in healthy controls. It is yet unknown what precise processes underlie this correlation and what clinical effects vitamin K insufficiency has on psoriasis [3,4].

The results of the literature review point to a tenable link between the pathophysiology of psoriasis vulgaris and vitamin K insufficiency. Because of its anti-inflammatory, antioxidant, and anti-calcification qualities, vitamin K may be able to alter important pathways in the pathophysiology of psoriasis. However, more research is needed to determine the therapeutic efficacy of vitamin K supplementation as well as the clinical importance of vitamin K shortage in psoriasis. Future studies should concentrate on clarifying the processes via which vitamin K affects the onset and course of psoriasis and assessing the effectiveness and safety of vitamin K supplementation as a treatment for psoriasis [5].

Conclusion

As a result of its impact on inflammation, oxidative stress, and tissue calcification, vitamin K deficiency contributes to the pathophysiology of psoriasis vulgaris. The incidence of vitamin K insufficiency in psoriasis and its clinical ramifications are not well documented, despite preclinical research suggesting that vitamin K may have anti-psoriatic properties. To further understand how vitamin K contributes to the pathogenesis of psoriasis and assess the therapeutic potential of vitamin K supplementation in the treatment of psoriasis, more research is necessary. We find new ways to improve patient outcomes and optimize psoriasis treatment by filling in these information gaps.

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Conflict of Interest

There are no conflicts of interest by author.

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