

Role of antioxidants in Psoriasis – a long road ahead?

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Much research has been done on the pathogenesis of psoriasis during the last few decades. This has led to the development of several new strategies for its treatment. Psoriasis, like many other skin diseases, is increasingly viewed as a manifestation of dysregulated immunity and inflammation. There are several players in the pathogenesis of psoriasis including genes, cells, such as lymphocytes, dendritic cells, mast cells, neutrophils and keratinocytes, cytokines, such as interferon alpha and gamma, interleukins 17, 22 and 23, tumour necrosis factor-alpha, innate immunity and autoantigens.¹ Two other factors of current interest are oxidative stress and the altered expression of microRNAs by the keratinocytes.^{2,3}

Cells use several methods to preserve intracellular levels of reactive oxygen species (ROS) and overall redox homeostasis to avoid damage to DNA, proteins and lipids.² Oxidative stress is a state of imbalance wherein reactive oxygen species overwhelm the intrinsic antioxidant mechanisms resulting in cellular damage. It is known to have a role in the pathogenesis of various diseases like hypertension, diabetes mellitus, heart disease, cancers and skin diseases like atopic dermatitis, seborrhoeic dermatitis, vitiligo, psoriasis, alopecia areata, lichen planus, acne and rosacea.²

Previous studies have demonstrated that the total oxidant capacity is high in psoriasis, whereas the total antioxidant capacity is low.⁴ The ischaemia-modified albumin (IMA) which is a marker of oxidative stress is also increased.⁵ Expression of micro-RNA (mi R146a and mi R203) is altered on lesional keratinocytes and peripheral blood.³ In this issue of the journal, Uzun *et al.* from Turkey report the results of their research concerning these processes.⁶ They have concluded that the total antioxidant capacity is markedly decreased

in patients with psoriasis. The decrease in the levels of ischaemia-modified albumin and increased expression of mi R203 noted in univariate analysis did not remain significant in multivariate analysis. Moreover, the finding of decreased ischaemia-modified albumin is not consistent with previous studies and is against the conventional pathogenetic model of the disease.

The possible effect of treatment on the variables and not measuring serum albumin which could affect ischaemia-modified albumin have been mentioned as limitations of the study by the authors. There could be a few more. For instance, it may not be possible to attribute an aetiologic role for the biochemical changes mentioned in this study. This is because it is difficult to ascertain if these preceded the onset of psoriasis. The sample size has been calculated based on the prevalence of psoriasis and not on any of the exposure or outcome variables of the study which would have been relevant here. Though the authors report the results of multivariate regression, it is not clear whether they considered confounding factors such as age, gender or obesity in the regression model.

Targeting oxidative stress has been an attractive strategy in the treatment of various skin diseases, including psoriasis. However, the available evidence is not sufficient to establish their efficacy.² Based on this report, it may be worthwhile to continue research on the role of these and other possible pathogenetic factors, some of which have a fundamental bearing on the development of psoriasis. Such research may also lead to the development of newer treatment modalities for psoriasis in future.

How to cite this article: Asokan N, Anjaneyan G. Role of antioxidants in Psoriasis-a long road ahead? *Indian J Dermatol Venereol Leprol* 2023;89:499–500.

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Received: May, 2023 **Accepted:** May, 2023 **Published:** June, 2023

DOI: 10.25259/IJDVL_569_2023 **PMID:** ****

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Declaration of patient consent

Patient's consent is not required as there are no patients in this study.

Financial support and sponsorship

Nil.

Conflict of interest

There is no conflict of interest.

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