

Functional approach to autoimmune skin diseases

Volume 10 Issue 1 - 2026

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Editorial

Understanding the molecular pathogenesis of autoimmune skin diseases could lead to the development of new, more effective therapeutic approaches with fewer adverse reactions, physiological but erroneous immune responses directed against skin pathogens are responsible for most inflammatory dermatoses (type 1 against viruses, type 2 against parasites, and type 3 against fungi and bacteria). Since the beginning of dermatology, therapies for these diseases have been acquired through trial and error or by anticipating the beneficial effects of products developed for other skin conditions.¹⁻³

Today, the focus is on a more functional approach to therapeutic interventions for skin diseases, one expressly aimed at combating them specifically that is, *targeted therapy*, this approach stems from a greater understanding of the molecular mechanisms involved in different autoimmune skin diseases (specific cell types, cytokines and their receptors, and inflammatory pathways as a whole), because the immune pathways that are dysregulated in the genesis of different dermatoses are distinct, resulting from the inappropriate activation of specific segments of the immune system.^{2,4}

Given their complexity, autoimmune skin diseases do not fit into general immune response models, but rather into a composition of different patterns, since they involve the dermis, epidermis, subcutaneous tissue, and blood vessels, in addition to changes in the pathophysiological patterns of skin lesions, for example, from blisters to lichenoid lesions. In this regard, under certain conditions, endogenous nucleic acids are perceived by the immune system as foreign molecular patterns, activating analogous immune pathways that trigger pro-inflammatory cascades. Similar mechanisms (in nucleic acid expression products) have been described against proteins of the dermal-epidermal adhesion complex.^{2,5,6}

This knowledge of the immune pathophysiology of autoimmune diseases is allowing the functional targeting of treatment towards the inhibition of key proteinases and cytokines involved that are overexpressed in individual diseases and even in the respective family of disorders related to the type of immune response targeted by the pharmacological action.² Finally, the functional approach to the treatment (targeted and stratified) of autoimmune skin diseases, based on the interpretation of the underlying pathophysiological mechanisms, especially those that have been little investigated, will allow for the efficient and precise treatment of those affected.

Acknowledgments

None.

Conflicts of interest

The author declares there is no conflict of interest.

Funding

None.

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