

Dermatitis And Eczema

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Abstract

Dermatitis is a common chronic inflammatory and immune-mediated skin disease with a complex pathophysiology and still represents a therapeutic challenge, owing to limited responses to available treatments. However, recent advances in the understanding of dermatitis pathophysiology have led to the discovery of several new potential therapeutic targets, and research in the field of new molecules with therapeutic perspectives is boiling, with more than 70 new promising drugs in development. The aim of this systematic review is to provide the state of the art on the current knowledge concerning the pathophysiology of the disease and on novel agents currently being investigated for AD, and to highlight which type of evolution is going to take place in therapeutic approaches of dermatitis in the coming years.

Keywords: dermatitis; pathophysiology; small molecules; biologics; novel treatments

Introduction

Eczema and dermatitis are terms used to describe the characteristic clinical appearance of inflamed, dry, occasionally scaly, and vesicular skin rashes associated with divergent underlying causes. The word eczema is derived from Greek, meaning ‘to boil over’, which aptly describes the microscopic blisters occurring in the epidermis at the cellular level. Dermatitis, as the term suggests, implies inflammation of the skin which relates to the underlying pathophysiology

It is one of the most common inflammatory disorders, affecting up to 20% of children and 10% of adults in high-income countries.^{1, 2} Globally, the prevalence of atopic dermatitis is increasing, although estimates in high-income countries are stabilising.

In this Article, we aim to discuss major developments in the understanding of dermatitis causes and long-term outcomes and to summarise the rapid expansion of therapeutic options, including targeted therapies.

"Materials and methods"

This systematic review was conducted following the approach developed by Arksey and O’Malley [3,4], which consists of five key steps: the identification of the research question; the identification of adequate studies; the selection of studies; the monitoring of data; and the collection, summary and reporting of results. The Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) extension for scoping review criteria was used to conduct the review.

Pathophysiology

The underling driver for atopic dermatitis (AD) is genetic predisposition (personal/family history of atopy). In those with AD there is an imbalance of T-helper lymphocytes, leading to increasing numbers

of Th-2 cells compared to Th-1 and Th-17. The abnormal Th-2 cells interact with Langerhans cells, causing raised levels of interleukins/immunoglobulin E (IgE) and a reduction in gamma interferon with resultant upregulation of pro-inflammatory cells. In addition to this, immune dysregulation gene defects have been identified in large cohorts of patients with AD including filaggrin gene defects which lead to impaired skin barrier function. This impaired barrier leads to increased transepidermal water loss and increased risk of antigens and infective organisms entering the skin.

Conclusion

Dermatitis is a common inflammatory skin disorder characterised by recurrent eczematous lesions and intense itch. The disorder affects people of all ages and ethnicities, has a substantial psychosocial impact on patients and relatives, and is the leading cause of the global burden from skin disease. Dermatitis is associated with increased risk of multiple comorbidities, including food allergy, asthma, allergic rhinitis, and mental health disorders. The pathophysiology is complex and involves a strong genetic predisposition, epidermal dysfunction, and T-cell driven inflammation. Although type-2 mechanisms are dominant, there is increasing evidence that the disorder involves multiple immune pathways. Currently, there is no cure, but increasing numbers of innovative and targeted therapies hold promise for achieving disease control, including in patients with recalcitrant disease.

Reference

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