Immune responses and therapeutic options in psoriasis

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Abstract

Psoriasis is a chronic inflammatory disease of the skin that affects about 2–3% of the population and greatly impairs the quality of life of affected individuals. Psoriatic skin is characterized by excessive proliferation and aberrant differentiation of keratinocytes, as well as redness caused by increased dilation of the dermal blood vessels and infiltration of immune cells. Although the pathogenesis of psoriasis has not yet been completely elucidated, it is generally believed to arise from a complex interplay between hyperproliferating keratinocytes and infiltrating, activated immune cells. So far, the exact triggers that elicit this disease are still enigmatic, yet, it is clear that genetic predisposition significantly contributes to the development of psoriasis. In this review, we summarize current knowledge of important cellular and molecular mechanisms driving the initiation and amplification stages of psoriasis development, with a particular focus on cytokines and emerging evidence illustrating keratinocyte-intrinsic defects as key drivers of inflammation. We also discuss mouse models that have contributed to a better understanding of psoriasis pathogenesis and the preclinical development of novel therapeutics, including monoclonal antibodies against specific cytokines or cytokine receptors that have revolutionized the treatment of psoriasis. Future perspectives that may have the potential to push basic research and open up new avenues for therapeutic intervention are provided.