IMMUNOBIOLOGICAL ASPECTS OF PSORIASIS PATHOGENESIS

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Relevance. Psoriasis is a chronic, immune-mediated inflammatory skin disease characterized by disrupted keratinocyte proliferation and differentiation, activation of innate and adaptive immunity, and the establishment of a persistent inflammatory process. Current research highlights the pivotal role of the interleukin-23 (IL-23)/interleukin-17 (IL-17) immune axis in the disease's pathogenesis. Investigating the molecular and cellular mechanisms of psoriasis is crucial for understanding its chronic, relapsing course and for developing targeted therapies.

Objective. To summarize current scientific data on the immunobiological mechanisms underlying psoriasis development, focusing on the interplay between innate and adaptive immune cells, the role of pro-inflammatory cytokines, and the mechanisms sustaining chronic inflammation.

Materials and Methods. An analytical review of international scientific publications regarding the immunopathogenesis of psoriasis was conducted, covering research on cellular inflammatory mechanisms, cytokine regulation, and intercellular interactions. The review was primarily based on studies by Sieminska et al. (2024) and Kamata and Tada (2023), as well as publications concerning the immune microenvironment of psoriatic skin. A comparative analysis and systematization of the published data were performed. No original clinical or experimental studies were conducted.

Results. Analysis of the scientific literature indicates that the development of psoriasis results from the interplay of genetic predisposition, environmental factors, and immune dysregulation.

Epidermal injury triggers the release of the antimicrobial peptide LL-37, which forms complexes with endogenous nucleic acids and activates plasmacytoid dendritic cells via Toll-like receptors TLR7 and TLR9. The subsequent production of type I interferons promotes the activation of myeloid dendritic cells and the establishment of an inflammatory microenvironment. The IL-23/Th17/IL-17 signaling axis plays a central role in sustaining psoriatic inflammation. Interleukin-23, produced primarily by activated dendritic cells, supports the survival and pathogenic activity of Th17 lymphocytes by stimulating the production of IL-17A, IL-17F, and IL-22. These cytokines act on keratinocytes, inducing excessive proliferation, impaired terminal differentiation, and enhanced expression of proinflammatory mediators. IL-17 activates intracellular NF-κB and MAPK signaling pathways, thereby increasing the synthesis of chemokines (CXCL1, CXCL8, CCL20), antimicrobial peptides, and IL-36. This facilitates the recruitment of neutrophils and other immunocompetent cells to the site of inflammation. Tumor necrosis factor-alpha (TNF-α) amplifies the effects of IL-17, creating a positive feedback loop between immune cells and keratinocytes that drives the chronicity of the inflammatory process. Tissue-resident memory T cells (Trm) play a crucial role in the relapsing nature of the disease, persisting in the skin even after the clinical resolution of psoriatic lesions. Their reactivation is accompanied by the production of pro-inflammatory cytokines and may contribute to the recurrence of inflammation in previously affected skin areas. The established role of the IL-23/IL-17 axis has provided the pathogenetic rationale for the use of biological agents designed to selectively inhibit key pro-inflammatory cytokines.

Conclusion. Psoriasis is a multifactorial, immune-mediated disease rooted in disrupted interactions between innate and adaptive immunity. The IL-23/Th17/IL-17 signaling axis plays a pivotal role in the development and maintenance of chronic inflammation by driving keratinocyte activation and amplifying

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the cytokine cascade. The persistence of tissue-resident memory T cells is considered a mechanism underlying disease recurrence. Further research into the immunobiological mechanisms of psoriasis offers prospects for refining personalized targeted therapy and achieving long-term clinical remission.

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