TLR3 ACTIVATION OF KERATINOCYTES INDUCES SKIN BARRIER REPAIRMENT IN ATOPIC DERMATITIS PATIENTS

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Introduction: Atopic dermatitis (AD) is a common immune-mediated chronic skin disease. Alteration of the lipid composition (abnormal ceramide profiles and lamellar lipid organization), as well as the decreased expression of the tight junction and cell structure proteins (claudin-1, filaggrin) are well-known. Since previous studies suggested that the stimulation of keratinocytes via the TLR3 pathway may improve the skin barrier repair, our aim was to investigate the impact of TLR3 activation of keratinocytes from AD patients regarding the expression of genes involved in lipid and in tight junction composition.

Methods: Outer root sheath keratinocytes from healthy controls and AD patients were cultured in our study, and 1ug/ml Poly(I:C) was used to activate the TLR3 pathway in the keratinocytes. After the first passage we used qPCR to measure the gene expression levels.

Results: In our results, the basic expression of genes involved in lipid composition (SMPD1, ABCA12 and GBA) and in the formation of skin barrier (CLDN1, IL-22R) was significantly altered in AD patients compared to healthy controls. However, 24 hours long exposition of Poly(I:C) significantly elevated the expression of the above mentioned genes.

Conclusion: Summarizing our results, patients suffering from AD have mRNA-level alterations in lipid composition as well as in tight junction formation, although this downregulation of the genes can be normalized with the activation of TLR3 pathway. Our data are the first findings, which may indicate a beneficial role of TLR3-based treatments in AD patients to repair the skin barrier.

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