Study establishes IL-24 as a critical factor for onset and progression of atopic dermatitis

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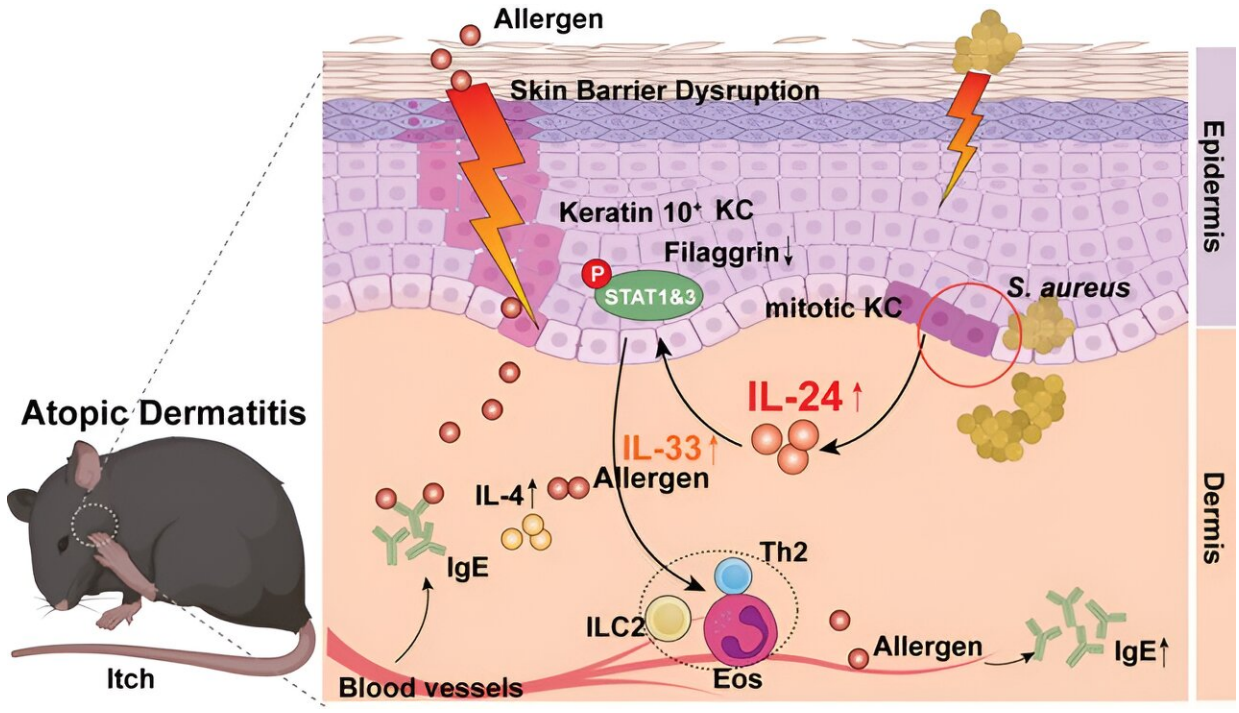

IL-24, which is expressed in keratinocytes and induced by S. aureus infection, promotes atopic dermatitis-like inflammation by enhancing a type 2 immune response. Credit: Xinmin Qian, Meiyi Tong, Tianqing Zhang, Qingqing Li, Meng Hua, Nan Zhou, and Wenwen Zeng

A study published in *Protein & Cell* details experiments conducted to understand the role of IL-24 in atopic dermatitis (AD)-like conditions.

The findings indicate that MRSA infection leads to increased IL-24 in keratinocytes, which in turn heightens type 2 immune responses and worsens AD symptoms. The paper, titled "IL-24 promotes atopic dermatitis-like inflammation through driving MRSA-induced allergic responses," explains the signaling pathways involved, particularly the JAK-STAT-IL-33 axis, and discusses the potential of targeting IL-24 for AD treatment.

Key findings from the study include:

1. IL-24 induction by MRSA: IL-24 is significantly induced in keratinocytes upon MRSA infection. This was revealed through single-cell RNA sequencing, showing a distinct immune response characterized by IL-24 upregulation.
2. Exacerbation of AD symptoms: Administration of recombinant IL-24 protein in animal models worsened AD-like pathology. Conversely, genetic deletion of Il24 or Il20rb in keratinocytes alleviated allergic inflammation and reduced AD symptoms.
3. IL-24 and IL-33 production: IL-24 acts through its receptors on keratinocytes to increase IL-33 production. Elevated IL-33 levels further promote type 2 immunity and exacerbate AD-like conditions.
4. Therapeutic potential: Blocking the JAK-STAT signaling pathway with Ruxolitinib reduced IL-33 levels induced by IL-24, indicating a potential therapeutic approach for AD. Specific deletion of Il20rb in keratinocytes also prevented increased IL-33 production.

This study identifies IL-24 as a crucial mediator in the development and exacerbation of AD-like inflammation, particularly in the presence of MRSA colonization. By promoting type 2 immune responses and increasing IL-33 production, IL-24 significantly worsens AD symptoms.

The findings suggest that targeting IL-24 or its signaling pathways could offer new therapeutic strategies for managing AD. The research highlights the complex interactions between microbial colonization, skin cell responses, and immune modulation, providing a deeper understanding of AD pathogenesis and potential avenues for intervention.

More information: Xinmin Qian et al, IL-24 promotes atopic dermatitis-like inflammation through driving MRSA-induced allergic responses, *Protein & Cell* (2024). DOI: 10.1093/procel/pwae030

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