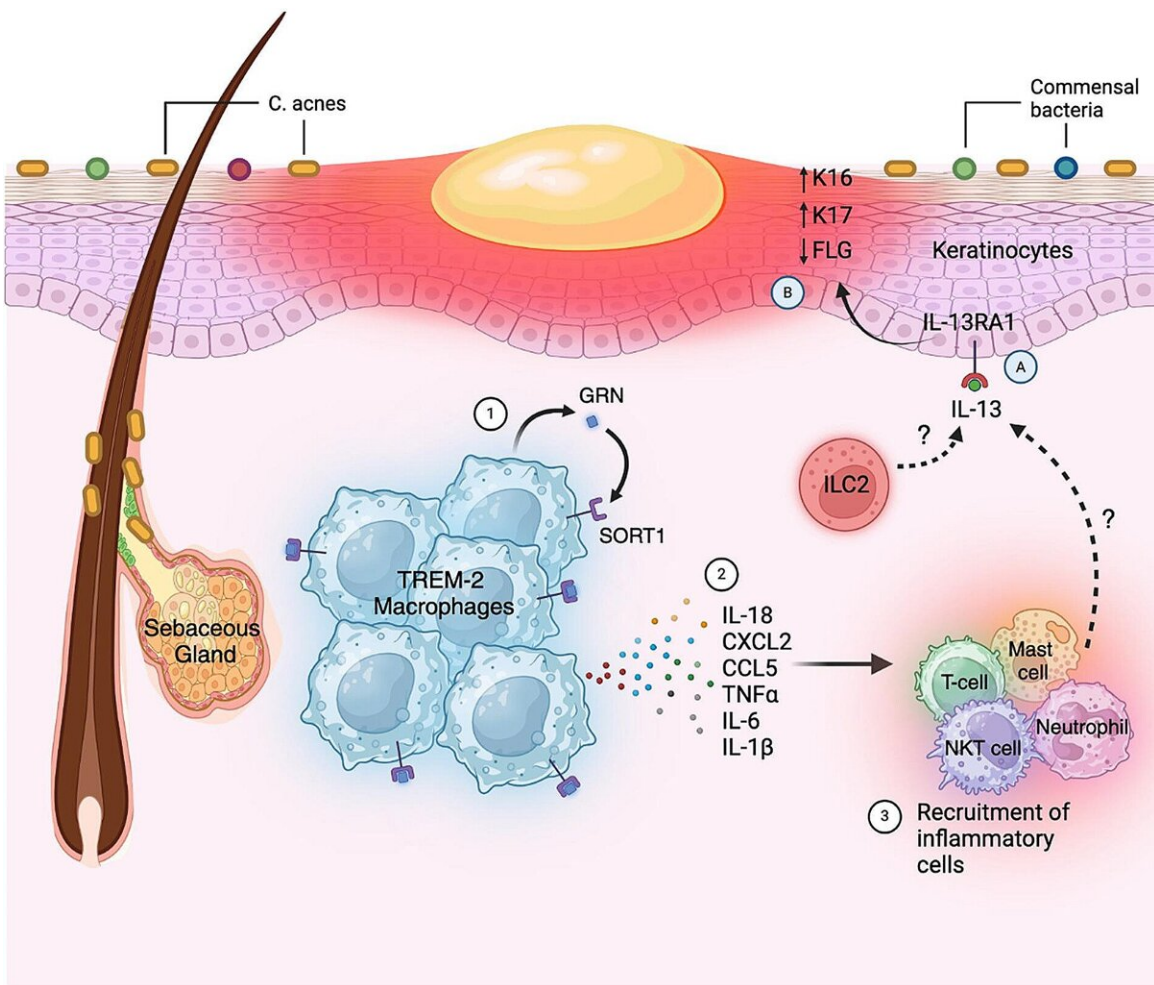


Researchers identify cellular pathways whose dysregulation leads to acne lesions

September 10 2024



A schematic overview highlighting the critical role of GRN in promoting inflammatory responses in TREM2 macrophages and IL-13RA1 in modulating the expression of genes associated with hyperkeratinization in acne skin. Credit: *Cell Communication and Signaling* (2024). DOI: 10.1186/s12964-024-01725-4

Acne is the most common dermatological condition worldwide and has been shown to have significant psychological effects on sufferers, especially teenagers.

While the condition involves excess production of lipids in the hair follicles, keratinocyte hyperkeratinization, bacterial interaction and a host of inflammatory responses involving [macrophages](#), little has been understood about the cascade of dysregulated signaling pathways—the cell-to-cell communication—that results in acne-affected skin.

Using cutting-edge bioinformatics techniques for gene identification and sequencing, Min Deng, Ph.D., in the George W. Agak Laboratory and colleagues at the David Geffen School of Medicine at UCLA are the first to comprehensively map signaling pathway distributions in both normal and acne-affected skin.

The research is [published](#) in the journal *Cell Communication and Signaling*.

Their analysis identified two key dysregulated pathways: GRN-SORT1 axis in TREM2 macrophages and the IL-13-IL-13RA1 axis in keratinocytes, both consistently upregulated in acne lesions. Specifically, their [comparative analysis](#) revealed changes in 49 individual pathways among seven different cell types in healthy versus affected skin.

The findings suggest that modulating these dysregulated signaling pathways through [gene therapy](#) could be a promising approach for the development of new [acne](#) treatments, especially those that target the early stage of lesion development.

More information: Min Deng et al, Analysis of intracellular communication reveals consistent gene changes associated with early-stage acne skin, *Cell Communication and Signaling* (2024). [DOI:](#)

[10.1186/s12964-024-01725-4](https://doi.org/10.1186/s12964-024-01725-4)

Provided by University of California, Los Angeles

Citation: Researchers identify cellular pathways whose dysregulation leads to acne lesions (2024, September 10) retrieved 10 September 2024 from

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