ACNE, ROSACEA, AND RELATED DISORDERS (INCLUDING HIDRADENITIS SUPPURATIVA)

TOLL-LIKE RECEPTOR SIGNALING PATHWAY, TH17 CELL DIFFERENTIATION AND DEFENSE RESPONSE TO MICROBES TAKE PART IN ACNE FLARE-UP IN TREATMENT PROCESS

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Background: Recent studies have found that gene expression changes were related to acne vulgaris. But the gene expression changes in acne flare-up after treatment is still unclear.

Objective: To identify gene expression changes in acne flare-up patients, thereby exploring the mechanisms of acne flare-up after treatment.

Material and methods: Eleven acne patients and three healthy people were include and divided into four groups (Group1: 4 with flare-up, Group2: 4 with improvement, Group3: 3 without obvious changes, Group4: healthy control). Peripheral blood of before and after isotretinoin or minocycline were collected. RNA-seq were used to detect the gene expression. We applied data in self-contrast and intergroup comparisons. Enrichment analysis based on the DEGs were performed via Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) database. Quantitative real-time PCR were then performed to confirm the RNA-seq results.

Results: Twenty-two genes were significantly upregulated after treatment compared to before in acne flare-up patients , which were involved in Toll-like receptor signaling pathway, NF-Kb signaling pathway, innate immune response and inflammatory response. Between flare-up group and without obvious changes group 1778 genes expression were upregulated and were enriched in Th17 cell differentiation, Th1 differentiation and NF-KB signaling pathway, and 57 genes expression were downregulated and were enriched in defensive response to organism, membrane disruption in other organisms, antimicrobial humoral response.

Conclusions: The gene expression profiles of acne flare-up patients changed. Inflammatory, immune responses play a prominent role in acne flare-up process and relatively weak defensive response to microbes, comedogenesis may be risk factors.