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Genome Wide Transcriptome Analysis of IL-17 and STAT3 Loop Activation Psoriasiform Murine Model

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Psoriasis is an autoimmune skin disease characterized by T lymphocytes activation and keratinocytes excessive proliferation and abnormal differentiation. Recently, T-helper 17 (Th17) cells and its related cytokines have been important influence factors in the pathology of psoriasis. Signal transducers and activators of transcription 3 (STAT3) over expression mice representing for keatinocytes proliferation. We tried to apply IL-17 (2μg / mouse) intradermal injection on STAT3 mice for 24 hours forming psoriasis-like lesions. The experiment was divided into three groups: wild-type (C57BL/6) mice, STAT3 mice and IL-17-treated STAT3. Illumumia sequence technique was applied to the dorsal skin of three groups (n=3). The results showed that 203 up regulated and 75 down regulated mRNAs were identified in IL-17A treated STAT3 mice compared to WT mice. The up-regulated included the members of S100A protein family such as S100A8 and S100A9, small proline-rich protein 2 (Sprr2) such as Sprr2e, Sprr2g and Sprr2d, late cornified envelope-3 (LCE) genes such as LCE3d, LCE3e and LCE3f. Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analysis dispicted the De-genes annotated pathway including innate immune response, arachidonic acid metabolism, cytokine-cytokine receptor interaction, *et al.* Those were in accordance with the pathological characteristics of psoriatic patients. In conclusion, our study verified that IL-17 and STAT3 loop activation play a key role in the pathogenesis of psoriasis, and IL-17-treated STAT3 mice model could be used as an animal model for the study of psoriasis.

Biography:

Xinran Xie is on-job doctorate, assistant researcher. The main research direction is the study of pathogenesis in psoriasis.