

A new ERA for global Dermatology 10 - 15 JUNE 2019 MILAN, ITALY

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

## THERAPEUTIC EFFECTS OF SALICYLIC ACID ON ACNE VULGARIS IN HUMAN SEB-1 SEBOCYTES ARE ASSOCIATED WITH REGULATION OF SREBP-1 PATHWAY AND NF-KB PATHWAY.

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Introduction: Acne vulgaris is a prevalent cutaneous disease characterized by multifactorial pathogenic processes including hyperseborrhea, inflammation, hyperkeratinization of follicular keratinocytes and Propionibacterium acnes (P. acnes) overgrowth. Salicylic acid (SA), a beta-hydroxy acid, is frequently used in acne treatment. It was reported that SA decreases skin lipids and has anti-inflammatory properties, however, few studies concentrate on the mechanisms and the exact pathways involved are still unclear.

Objective: This study aims to investigate the therapeutic effects of SA on acne vulgaris in human SEB-1 sebocytes.

Materials and Methods: The SEB-1 human sebocyte cell line was cultured in vitro. The rabbit ear acne model was established by topical application of coal tar. Gas chromatography-mass spectrum analysis was used to determine lipid content in human SEB-1 sebocytes. Inflammatory factors in medium were determined by ELISA assay. RT-qPCR and western blot analysis were used to determine the gene expressions. Flow cytometry was performed to evaluate cell apoptosis. Immunohistochemical staining was performed to determine the level of major pathogenic proteins in rabbit ears acne lesions.

Results: SA decreased lipogenesis by downregulation of the adenosine monophosphate-activated protein kinase (AMPK)/sterol response element-binding protein-1 (SREBP-1) pathway and reduced inflammation by suppressing the NF-kB pathway in human SEB-1 sebocytes. Salicylic acid also decreased the viability of SEB-1 sebocytes by increasing apoptosis through the death signal receptor pathway. Histopathological analysis of rabbit ear acne model after applying SA for 3 weeks confirmed that SA decreased the levels of cytokines and major pathogenic proteins around acne lesions, which provides solid evidence for suggested therapeutic mechanisms.

Conclusions: Our study initially provided evidence indicating that therapeutic effects of SA on acne vulgaris could be associated with the regulation of SREBP-1 pathway and NF-κB pathway in human SEB-1 sebocytes, which provided a novel insight for the therapeutic











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rationale of acne vulgaris.





