PSORIASIS

ONCOSTATIN M REGULATED SENESCENCE-AUTOPHAGY INTERACTION IN KERATINOCYTE IN THE PATHOGENESIS OF PSORIASIS

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Background: Psoriasis is a common chronic inflammatory skin disease characterized by abnormal keratinocyte proliferation and terminal differentiation. However, the etiology of psoriasis is not fully understood. In our previous studies, we found that Oncostatin M (OSM) was significantly overexpressed in psoriasis lesions and related to the expression of typical cellular senescence markers and autophagy markers.

Objective: To estimate the influence of Oncostatin M on the senescence-autophagy interaction in M5-induced HaCAT keratinocytes and human primary keratinocytes.

Materials: and methods OSM synthesis and expression of the OSM-related genes were analyzed by using RNA-seq quantitative reverse transcriptase polymerase chain reaction, immunostaining and Western blotting from normal and psoriatic lesion skin samples. The pharmacodynamics and mechanism of OSM inhibitor was evaluated in human keratinocyte and murine psoriasis models.

Rusults: Our current study demonstrates the OSM inhibitor can significantly inhibit the secretion of cytokines and psoriasis markers in M5-induced HaCAT keratinocytes and human primary keratinocytes, increasing the level of autophagy and decreasing the level of cellular senescence markers.

Conclusions: We speculate that the Oncostatin M regulated senescence-autophagy interaction in keratinocyte is related to the maintenance of stability of epidermal cells and skin barrier, thus playing an important role in the pathogenesis of psoriasis. In summary, our findings show that OSM is a key mediator in psoriatic dermatitis and might be an interesting therapeutic target in psoriasis patients.