



AESTHETIC AND COSMETIC DERMATOLOGY (LASERS SEPARATE CATEGORY)

GALACTOMYCES FERMENTATION FILTRATE PROTECTS HUMAN KERATINOCYTES AGAINST OXIDATIVE INDUCED-CELLULAR SENESCENCE VIA INDUCTION OF AUTOPHAGY.

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Background: Our previous study has shown that Galactomyces fermentation filtrate (GFF), a cosmetic compound, is capable of activating aryl hydrocarbon receptor (AHR) in human keratinocytes. Because AHR has been reported to regulate autophagy, a self-digestion system for the intracellular components, we hypothesized that GFF may induce autophagy in human keratinocytes. Also, because autophagy has an important role in the prevention from cellular senescence, we aimed to investigate the effect of GFF-induced autophagy on cellular senescence caused by oxidative-stress.

Objective: To examine whether GFF protects human keratinocytes against oxidative induced-cellular senescence via induction of autophagy.

Materials and Methods: Normal epidermal human keratinocytes (NHEKs) were treated with or without GFF. The induction of autophagy was examined by western blotting (WB) analysis and immunofluorescence study. Next, NHEKs were exposed to H₂O₂ with or without GFF. The protein expression of CDKN2A, p53 and p21, markers of cellular senescence, was evaluated by WB analysis. AHR or control knockdown was carried out by transfection with AHR or control small interfering RNA.

Results: GFF treatment induced phosphorylation of AMPK and subsequently upregulated expression of LC3B, a central protein in the autophagy pathway. GFF treatment also inhibited expression of SIRT3, a negative regulator of autophagy, which was cancelled by siRNA transfection of AHR. The GFF treatment inhibited H₂O₂-induced CDKN2A, p53 and p21, which was reversed by siRNA transfection of AHR and ATG5, a protein specifically required for autophagy.

Conclusions: GFF is useful to prevent cellular senescence in an AHR-mediated autophagy fashion.

