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The bitter taste receptor agonists amarogentin and Gentiana lutea extract modulate cell differentiation and lipid synthesis in keratinocytes

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We have recently shown that human keratinocytes express the human bitter taste receptors TAS2R1 and TAS2R38. Here, we have analyzed if TAS2R agonists induce the synthesis of skin barrier proteins and lipids in keratinocytes.

HaCaT keratinocytes were stimulated with the TAS2R agonist amarogentin, a characteristic bitter agent of Gentiana lutea extract (GE). Amarogentin induced calcium influx and promoted the expression of keratin 10, involucrin and transglutaminase.

Furthermore we analyzed if GE additionally has an effect on lipid synthesis in keratinocytes. To address this issue, we performed a quantitative fluorescence assay with the dye Nile Red that is only fluorescent in a hydrophobic environment. Primary keratinocytes were incubated for 6 days with GE. Nile Red labeling revealed that GE significantly increased lipid synthesis in keratinocytes. No toxic effects of amarogentin and GE could be detected.

Because epidermal proteins and lipids are essential for building an intact epidermal barrier, amarogentin and GE might be used to improve skin conditions with an impaired barrier function.