

# **Biochemical investigation and gene analysis of equol: A plant and soy-derived isoflavonoid with antiaging and antioxidant properties with potential human skin applications.**

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Article first published online: 27 JAN 2012

## **Keywords:**

- equol;
- extracellular matrix;
- gene expression;
- human skin

## **Abstract**

The purpose of this study was to investigate the effects of equol, a plant and intestinal flora derived isoflavonoid molecule on the expression of skin genes and proteins using human dermal models. As equol has been shown to mimic  $17\beta$ -estradiol and bind specifically to  $5\alpha$ -dihydrotestosterone ( $5\alpha$ -DHT), these agents were used (in addition to equol) to determine whether equol may play important and beneficial roles in the extracellular matrix (ECM). Equol at 0.3 or 1.2% in qPCR experiments using a human skin barrier model examined ECM gene expression. Equol,  $5\alpha$ -DHT, and  $17\beta$ -estradiol at 10 nM were studied in human monolayer fibroblasts cultures (hMFC) for ECM protein expression. Human fibroblast three-dimensional organotypic cultures revealed equol's influence (@ 10 nM) on ECM proteins via fluorescent-activated cell sorting (FACS) analysis. In qPCR experiments, equol significantly increased collagen, elastin (ELN), and tissue inhibitor of metalloprotease and decreased metalloproteinases (MMPs) gene expression and caused significant positive changes in skin antioxidant and antiaging genes. In hMFC, equol significantly increased collagen type I (COL1A1), whereas,  $5\alpha$ -DHT significantly decreased cell viability that was blocked by equol. FACS analysis showed equol and  $17\beta$ -estradiol significantly stimulated COL1A1, collagen type III (COL3A1), and ELN while MMPs were significantly decreased compared with control values. Finally, tamoxifen blocked the positive influences of equol on ECM proteins via FACS analysis. These findings suggest that equol has the potential to be used topically for the treatment and prevention of skin aging, by enhancing ECM components in human skin.

## **How to Cite**

Gopaul, R., Knaggs, H. E. and Lephart, E. D. (2012), Biochemical investigation and gene analysis of equol: A plant and soy-derived isoflavonoid with antiaging and antioxidant properties with potential human skin applications. BioFactors. doi: 10.1002/biof.191