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Ac-SDKP suppresses epithelial-mesenchymal transition in A549 cells via HSP27 signaling

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The synthetic tetrapeptide N-acetyl-seryl-aspartyl-lysyl-proline (Ac-SDKP) has shown to be a modulator of molecular aspects of the fibrosis pathway. This study reveals that Ac-SDKP exerts an anti-fibrotic effect on human type II alveolar epithelial cells (A549), which are a source of myofibroblasts once exposed to TGF-β1, by decreasing the expression of heat shock protein 27 (HSP27). We used A549 cells in vitro to detect morphological evidence of epithelial-mesenchymal transition (EMT) by phase-contrast microscopy. Immunocytochemical and Western blot analysis determined the distributions of cytokeratin8 (CK8), α-smooth muscle actin (α-SMA), and SNAI1. Confocal laser scanning microscopy revealed a co-localization of HSP27 and SNAI1 on TGF-β1-induced A549 cells. These results also demonstrated that A549 cells became spindle-like when exposed to TGF-β1. Coincident with these morphological changes, expression levels of CK8 and E-cadherine decreased, while those of vimentin and α-SMA increased. This process was accompanied by increases in levels of HSP27, SNAI1, type I and type III collagen. In vitro transfection experiments demonstrated that the inhibition of HSP27 in cultured A549 cells could decrease the expression of SNAI1 and α-SMA while increasing the expression of E-cadherine. A noticeable reduction in collagen types I and III were also evident. Our results found that Ac-SDKP inhibited the transition of cultured A549 cells to myofibroblasts and attenuated collagen synthesis through modulating the expression of HSP27.

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Genetic variations associated with vitamin D (cholecalciferol) bioavailability

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Most people require dietary vitamin D (VD) to achieve the recommended blood level of 25-hydroxycholecalciferol (25OHD). However, blood response to VD supplementation is highly variable among individuals. Our main objective was to assess whether the variability in D_3 bioavailability was associated with single nucleotide polymorphisms (SNPs) in candidate genes. 39 healthy adult men were genotyped using whole-genome microarrays. Following an overnight fast, plasma 25OHD status was measured and the subjects then consumed a meal providing 5 mg D_3 as a supplement. Plasma chylomicron D_3 concentrations were measured over 8 h, and D_3 response calculated by determining the postprandial AUC. Partial least squares regression was used to assess the association of SNPs in or near candidate genes (61 genes representing 3791 SNPs) with the postprandial D_3 response. The mean postprandial chylomicron D_3 concentration peaked at 6 h. The D_3 response was very variable among individuals (CV=47%), and it did not correlate with the fasting plasma 25OHD concentration. A significant ($P=1.32x10-4$) partial least squares regression model, which included 17 SNPs in 13 genes were associated with the variance in the D_3 response. There is a high inter-individual variability in D_3 bioavailability which is associated with a combination of SNPs.

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