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Avocado/soya unsaponifiables enhance the expression of transforming growth factor beta1 and beta2 in cultured articular chondrocytes.

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OBJECTIVE: Avocado and soya unsaponifiables (ASU) have been reported to exert beneficial effects in the treatment of periodontal and osteoarticular diseases. They are supposed to stimulate deposition and repair of extracellular matrix components, but the mechanisms underlying their action are not well understood. In view of the repair potential of osteoarthritic (OA) cartilage and the role that the transforming growth factor beta (TGFbeta) system could play in that process, we carried out in vitro studies to determine the mechanism of action of ASU on articular chondrocytes that may account for the beneficial effects on cartilage metabolism. METHODS: Cultured bovine articular chondrocytes were treated with various concentrations of ASU, and the expression of both TGFbeta isoforms, 1 and 2, and their receptors (TGFbetaRI and TGFbetaRII) was determined by Northern blot and reverse transcriptase-polymerase chain reaction. Cell transfection with TGFbeta1 promoter constructs was also used to delineate the cis-acting sequences mediating ASU responsiveness in chondrocytes. The level of plasminogen activator inhibitor 1 (PAI-1) was also evaluated by Northern blotting and protein radiolabeling. RESULTS: The data indicated that ASU stimulate the expression of TGFbeta1, TGFbeta2, and PAI-1 by articular chondrocytes. In contrast, the levels of TGFbetaRI and TGFbetaRII were not significantly affected by the compound. Treatment of bovine articular chondrocytes transiently transfected with TGFbeta1 promoter constructs suggested that the effect on TGFbeta1 expression is mediated by the region located between -732 and -1132 bp. CONCLUSION: The results indicate that the ASU-induced stimulation of matrix synthesis previously reported in cultured articular chondrocytes could be explained by the ability to enhance TGFbeta expression in these cells. Further, ASU increase the production of PAI-1, an effect that could help in blocking the plasmin cascade that leads to metalloprotease activation. These data suggest that the compound has properties that might promote TGFbeta-induced matrix repair mechanisms in articular cartilage.

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