J Rheumatol. 2003 Aug;30(8):1825-34.

Avocado/soybean unsaponifiables increase aggrecan synthesis and reduce catabolic and proinflammatory mediator production by human osteoarthritic chondrocytes.

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OBJECTIVE: To investigate the effects of avocado (A)/soybean (S) unsaponifiables on the metabolism of human osteoarthritic (OA) chondrocytes cultured in alginate beads over 12 days.

METHODS: Enzymatically isolated OA chondrocytes were cultured in alginate beads in a well defined culture medium for 12 days, in the presence or not of 10-10 M interleukin 1beta (IL-1beta). DNA content was measured using a fluorometric method. Production of aggrecan (AGG), stromelysin-1 (MMP-3), tissue inhibitor of metalloproteinases-1 (TIMP-1), macrophage inflammatory protein-1beta (MIP-1beta), IL-6, and IL-8 were assayed by specific enzyme amplified sensitivity immunoassays. Prostaglandin (PG) E2 was measured by a specific radioimmunoassay and nitrite by a spectrophotometric method based on the Griess reaction. A commercial avocado and soybean mixture of unsaponifiables (A1S2) and each component separately were tested in a range of 0.625 to 40.0 micro g/ml.

RESULTS: After 12 days' incubation, A1S2 increased AGG synthesis and accumulation in alginate beads in a dose and time dependent manner. A1S2 promoted the recovery of aggrecan synthesis after 3 days of IL-1beta treatment. A1S2 was a potent inhibitor of basal and IL-1beta stimulated MMP-3 production. The procedure also weakly reversed the inhibitory effect of IL-1beta on TIMP-1 production. A1S2 inhibited basal production of MIP-1beta, IL-6, IL-8, NO*, and PGE2 by OA chondrocytes and partially counteracted the stimulating effect of IL-1 on PGE2. Compared to avocado or soybean added separately, the mixture had a superior effect on NO* and IL-8 production.

CONCLUSION: A1S2 stimulated aggrecan production and restored aggrecan production after IL-1beta treatment. In parallel, A1S2 decreased MMP-3 production and stimulated TIMP-1 production. These results suggest A1S2 could have structure-modifying effects in OA by inhibiting cartilage degradation and promoting cartilage repair.

PMID: 12913942 [PubMed - indexed for MEDLINE]