

Dietary fatty acids and immune reactions in synovial tissue.

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Abstract

Inflammation of the synovial membrane in rheumatoid arthritis is mediated by specialized cells necessary for response. The most prominent features are the accumulation of mononuclear phagocytes, lymphocytes and the proliferating tissue. Pro-inflammatory and proliferative signals are transmitted to the bone marrow and to the membrane. The result is a monoclonal stimulation of specific cell lines, and synovial proliferation in the inflamed membrane. Angiogenesis, synovial hypertrophy, and increased perfusion facilitate the accumulation of inflammatory cells. Components of the autoimmune reaction are described in the international system of classification, the CD-S (cluster of differentiation). Pro-inflammatory signals are mediated by metabolites of arachidonic acid. Prostaglandins, leukotrienes, lipoxins and hydroxy fatty acids, derived from this PUFA, stimulate the formation and the activation of adhesion molecules (integrins), cytokines (gamma-interferon, interleukin-1, interleukin-6, tumor-necrosis factor), chemokines (interleukin-8, macrophage-chemotactic peptide, RANTES and colony-stimulating factors (CSF, granulocyte/monocyte-CSF, Multi-CSF (= IL-3)). Dietary means to mitigate inflammation comprise reduction of arachidonic acid, and increased intake of eicosapentaenoic acid and antioxidants. In the literature 12 random placebo-controlled double-blind studies, fulfilling GCP-criteria, demonstrate a moderate but consistent improvement in clinical findings and laboratory parameters in patients with RA. A dose-response relationship was established with a daily dose of 2.6 gram fish oil, equivalent to about 1.6 gram EPA. In these experiments EPA was the omega-3 fatty acid responsible for improvement, with distinct effects on inhibition of cytokine formation (IL-1 to IL-6, IL-8, TNF- α , CSF), decreased induction of proinflammatory adhesion molecules (selectins, intercellular adhesion molecule-1), and degrading enzymes (e.g. phospholipase A2, cyclooxygenase-2, inducible NO-synthetase). Only one study reports the relevance of the background diet. From this study it became apparent that reduction of dietary arachidonic acid improves the incorporation and the clinical benefit of EPA.

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