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## **Green tea compound may be a therapy for people with rheumatoid arthritis, University of Michigan study finds**

### ***Anti-inflammatory compound inhibits production of molecules that cause destruction of cartilage and bone***

**ANN ARBOR, MI** – A new study from the [University of Michigan Health System](#) suggests that a compound in green tea may provide therapeutic benefits to people with rheumatoid arthritis.

The study, presented April 29 at the [Experimental Biology 2007](#) in Washington, D.C., looks at a potent anti-inflammatory compound derived from green tea. Researchers found that the compound – called epigallocatechin-3-gallate (EGCG) – inhibited the production of several molecules in the immune system that contribute to inflammation and joint damage in people with [rheumatoid arthritis](#).

The compound from green tea also was found to suppress the inflammatory products in the connective tissue of people with rheumatoid arthritis.

“Our research is a very promising step in the search for therapies for the joint destruction experienced by people who have rheumatoid arthritis,” says Salah-uddin Ahmed, Ph.D., lead researcher on the study. Ahmed, a research investigator with the [Division of Rheumatology](#) at the U-M Health System, was selected to present the research at the Experimental Biology meeting as the recipient of the Young Scientist Travel Award, given by the American Society for Pharmacology and Experimental Therapeutics. This study was also selected by the American Society for Nutrition to be featured in a press release.

To conduct the research, the scientists isolated cells called synovial fibroblasts from the joints of patients with rheumatoid arthritis. These fibroblasts – cells that form a lining of the tissue surrounding the capsule of the joints – then were cultured in a growth medium and incubated with the green tea compound.

The fibroblasts were then stimulated with pro-inflammatory cytokine IL-1b, a protein of the immune system known to play an important role in causing joint destruction in people with rheumatoid arthritis. The researchers looked at whether the green tea compound has the capability to block the activity of two potent molecules, IL-6 and cyclooxygenase-2 (COX-2), which also are actively involved in causing bone erosion in the joints of people with rheumatoid arthritis.

When untreated cells were stimulated with IL-1b, a sequence of molecular events occurred that resulted in production of the bone-destructive molecules. But the scientists found that pre-incubation with EGCG was capable of inhibiting the production of these molecules. EGCG also inhibited the production of prostaglandin E2, a hormone-like substance that causes inflammation in the joints.

The cell signaling pathways that regulate levels of these immune system molecules under both normal and rheumatoid arthritis situations are well studied, and the researchers were able to trace the effects of the green tea compound infusion to see that it worked by inhibiting these pathways.

Ahmed says that these studies suggest that EGCG or molecules that could be derived synthetically from the EGCG found in green tea may be of therapeutic value by inhibiting the joint destruction in rheumatoid arthritis.

Previously, Ahmed and other researchers made another promising finding when EGCG-pretreated synovial fibroblasts were stimulated with the cytokine IL-1b to study the protective effect of this green tea compound. Compared to untreated synovial fibroblasts, the cells treated with EGCG markedly blocked the ability of IL-1b to produce the proteins and enzymes that infiltrate the joints of persons with rheumatoid arthritis and cause cartilage degradation.

The laboratory now is focused on the inhibitory role of EGCG in gene expression. The scientists plan to test EGCG in animal models of rheumatoid arthritis to see if it provides similar therapeutic or preventive effects. Ahmed believes that the outcome of these studies will form a strong foundation for future testing of green tea compound in humans with rheumatoid arthritis.

In addition to Ahmed, authors of the study are Angela Pakozdi, M.D., a former research fellow in the Division of Rheumatology at the U-M Health System; and Alisa E. Koch, M.D., the Frederick G.L. Huetwell and William D. Robinson, M.D. Professor of Rheumatology at the U-M Health System and a researcher at the Veterans Affairs Ann Arbor Healthcare System.

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