New Clues to Rheumatoid Arthritis

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DENVER — National Jewish Medical and Research Center researchers have identified cells that contribute to inflammation and tissue damage in a mouse model of rheumatoid arthritis. The cells, known as gamma-delta T cells, produce IL-17, which is considered a major player in chronic autoimmune diseases. Depleting mice of these cells significantly lessened both the severity and incidence of disease. The results were published in the October 2007 issue of the *Journal of Immunology*.

“If we can manipulate gamma-delta T cells to produce less IL-17 we could potentially help rheumatoid arthritis patients,” said lead author, Christina Roark, PhD, Assistant Professor of Immunology at National Jewish. “These findings might also have implications for other inflammatory and autoimmune diseases.”

The researchers injected mice with collagen to induce a chronic inflammation that shares many of the hallmarks of rheumatoid arthritis. They found that a specific subset of gamma delta T cells, known as Vgamma4 cells, multiplied and became activated. The activated cells secreted the cytokine IL-17. When in the course of the disease, the researchers treated some of the mice with monoclonal antibodies that depleted their bodies of the Vgamma4 cells, those mice went on to develop significantly less inflammation and damage to cartilage than the mice having normal levels of the Vgamma4 cells. The research was funded by the Arthritis Foundation and the NIH.

The next step for researchers is to determine if whether a similar process occurs in humans with rheumatoid arthritis.

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