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Biomarker Could Help Doctors Tailor Treatment For Rheumatoid Arthritis

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Investigators have identified a biomarker that could help doctors select patients with rheumatoid arthritis who will benefit from therapy with drugs such as [Enbrel](#), a tumor necrosis factor (TNF)-antagonist drug. The study, led by researchers at Hospital for Special Surgery in collaboration with rheumatologists at University of Southern California, appears in the February issue of the journal *Arthritis & Rheumatism*.

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"While our study was performed on a relatively small group of patients and will need to be confirmed in a larger cohort, the data are promising and may be clinically significant for the medical management of patients," said Mary K. Crow, M.D., director of Rheumatology Research and co-director of the Mary Kirkland Center for Lupus Research at Hospital for Special Surgery. "Treatment with these drugs is very expensive; the drugs can cost around \$16,000 or so per year. If you are going to use them, you would like to know that they are likely to work in your patient." Other well-known TNF-antagonists include [Humira](#) and [Remicade](#).

While TNF antagonists have brought relief to thousands of people with rheumatoid arthritis, the drugs are not highly effective in 30 percent to 50 percent of patients. Clinicians thus run the risk of providing a therapy to patients that doesn't work well, is expensive and is potentially toxic. Patients taking TNF antagonists, which have been available for roughly ten years, can run the risk of developing bacterial or fungal infections.

While studies have identified factors associated with poor response to these drugs such as expression of certain genes, none of the factors has as yet provided doctors with a tool that will help select patients who are likely to respond to the drugs or identify those less likely to respond. Investigators at HSS hoped to remedy this and turned their attention to the type I interferon proteins, specifically a type called interferon beta (IFN-beta). Previous studies have revealed that levels of IFN-beta, a protein that can limit cell division, is present in the joint tissue of some patients with rheumatoid arthritis. The researchers wondered if variable levels of this protein could play a role in how patients respond to TNF-antagonist drugs. To test this hypothesis, the investigators set out to determine the relationship between levels of type I interferon activity in the blood prior to beginning therapy and the ability of the drug to control rheumatoid arthritis in patients. They studied the role of IFN-beta, and because they knew that IFN-beta induces interleukin-1 receptor antagonist (IL-1Ra), another protein, they also tested levels of IL-1Ra.

The study involved three cohorts of patients: patients who had rheumatoid arthritis and received a TNF antagonist (n=35), arthritis patients who received no drug (12), and healthy volunteers (n=50). Patients received their care at the Los Angeles County and University of Southern California Medical Center Rheumatology Clinics.

Outcomes were evaluated during a window of therapy consisting of more than three months but fewer than nine months, allowing for sufficient time for clinicians to determine clinical response. Doctors used a tool commonly employed to gauge the severity of arthritis—the Disease Activity Score in 28 joints—to deem whether patients had a moderate, good, or no response to the drug.

The investigators found that patients with higher baseline levels of type I IFN were

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"We have drawn attention to a potential biomarker that, if our results are supported by additional future studies in other patient populations, might provide a tool to predict who might be a responder to this class of biologic rheumatoid arthritis therapies, the TNF antagonists, and who might be less likely to be a responder," Dr. Crow said. "For those who demonstrate low levels of blood interferon activity, that information might be useful to guide patients to alternative treatments that might be more likely to work for them." This could include the use of other drugs such as Rituximab, which is not a TNF antagonist.

Dr. Crow was recently named physician-in-chief and chair of the Division of Rheumatology at Hospital for Special Surgery. This appointment is effective as of April 1.

Other authors of the study are Clio P. Mavragani, M.D., at Hospital for Special Surgery, and Dan T. La, M.D., and William Stohl, M.D., Ph.D., at the Los Angeles County and University of Southern California Keck School of Medicine, Los Angeles. Dr. Mavragani's work was supported by a Stavros Niarchos Fellowship from the New York Chapter of the Arthritis Foundation. Dr. Crow's work was supported by a grant from the National Institutes of Health, the Alliance for Lupus Research, the Lupus Research Institute, and the Mary Kirkland Center for Lupus Research at Hospital for Special Surgery.

Source
Hospital for Special Surgery

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