

Biomarkers of Chronic Fatigue Syndrome

Evidence could lead to test for mysterious condition



Stanford's Dr. Jose Montoya and his team with the Chronic Fatigue Initiative run a clinic that evaluates patients with chronic diseases in which infection or its immune response may play a role.

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PUBLISHED: July 31, 2017 at 12:03 pm | UPDATED: August 1, 2017 at 5:00 am

A new Stanford study has identified biomarkers linked to the severity of chronic fatigue syndrome, offering evidence that inflammation is a powerful driver of this mysterious disease, long dismissed as a psychological condition.

The team, led [Dr. Jose Montoya](#) of Stanford University School of Medicine, found that varying concentrations of 17 immune-system signaling proteins, or cytokines, in the blood correlate with the disease's severity.

The underpinnings of the disease have eluded researchers for 35 years. Patients experience debilitating fatigue, often unable to walk, talk or even eat.

Finding biomarkers for chronic fatigue syndrome has been an ongoing goal for researchers who hope to one day develop a diagnostic test for the condition, which affects an estimated 836,000

to 2.5 million Americans. Biomarkers are important because, unlike subjective symptoms, they offer an objective and measurable sign of a medical state.

“There’s been a great deal of controversy and confusion” surrounding the condition, said senior author Mark Davis, professor of immunology and microbiology and director of Stanford’s Institute for Immunity, Transplantation and Infection, in a prepared statement about the findings, published Monday in the [Proceedings of the National Academy of Sciences](#). “Our findings show clearly that it’s an inflammatory disease and provide a solid basis for a diagnostic blood test,” he said.

Montoya’s team enlisted Stanford’s [Human Immune Monitoring Center](#) to help. Founded a decade ago, the center is the engine for large-scale data-driven analyses of biological patterns in blood and tissue.

“His team’s discovery is a giant step forward,” said Esther Siebert, a member of the South Bay ME/CFS Support Group who has been living with the condition for 32 years. Before becoming sick, the University of Chicago graduate worked in marketing and sold large computer systems to Fortune 500 companies.

Urging the federal government to use these findings to expand research, Siebert urged that National Institutes of Health to boost funding “to replicate this study, develop biomarker tests and, hopefully in time, treatments.”

Many, but not all, patients experience flu-like symptoms common in inflammation-driven diseases.

Antivirals, anti-inflammatories and immune-modulating drugs have led to improvement in some cases. The new study holds implications for the direction of future studies, suggesting clinical trials to test the effectiveness of drugs that modulate the immune system.

The Stanford team analyzed blood samples from 192 Stanford patients as well as from 392 healthy control subjects.

It found that some cytokine levels were lower in patients with mild forms of the disease than in the control subjects, but elevated in patients with relatively severe manifestations. This may reflect different genetic predispositions, among patients, to progress to mild versus severe disease, the scientists believe.

Thirteen of these 17 cytokines are involved in inflammation.

Just as important, the study design separated patients into separate categories depending on disease severity. If the results had been simply averaged, the phenomenon might have been completely obscured.

A 2015 [Institute of Medicine report](#) concluded that it is an under-funded and under-researched disease. It urged a re-naming of the condition to Myalgic Encephalomyelitis/Chronic Fatigue Syndrome, to better reflect the seriousness of the condition.

Montoya is one of a handful of doctors across the country conducting promising cutting-edge research into the causes of and treatments for chronic fatigue syndrome. He leads Stanford's [Chronic Fatigue Initiative](#), which studies the roles that infection and the immune response play in the symptoms of patients suffering from the condition and other infection-associated chronic and unexplained diseases.

Much as different organisms can cause pneumonia, Montoya and his team believe that multiple pathogens, including viruses, can wreak havoc on the immune system, triggering chronic fatigue syndrome. But the precise triggers have not yet been identified.

"The beloved Dr. Montoya risked his career when the suffering of his first ME/CFS patient moved him to work on it," said Siebert.

"I am writing this through 'brain fog,'" she said. "We long-timers no longer fight for ourselves but for the youngsters confined to darkened rooms, unable to talk and being tube fed."



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