

# Chronic Fatigue Syndrome: Progress Toward Diagnosis and Treatment, Finally?



By Henry Miller — September 16, 2019



Is This You? Photo: Wikipedia Commons

Some medical conditions are especially frustrating to physicians because they lack not only effective treatments but even a reliable means of diagnosis. One of the most common of these is variously known as myalgic encephalomyelitis or [chronic fatigue syndrome \(ME/CFS\)](#) [1]. As the name suggests, it is a *syndrome*, a group of symptoms that seem to characterize, or define, an illness, even if we don't know how they're related or what causes them. That's different from better-understood illnesses such as solid tumors, rheumatoid arthritis, or stroke, for example.

The signs and symptoms may include fatigue, loss of memory or concentration, sore throat, swelling of neck or armpit lymph nodes, unexplained muscle or joint pain, headaches, unrefreshing sleep, and extreme exhaustion lasting more than 24 hours after physical exercise or mental stimulation ("post-exertional malaise"). People with ME/CFS are often incapable of performing ordinary activities, and sometimes become completely debilitated, unable even to get out of bed. The symptoms can persist for years.

The cause(s) of ME/CFS is not known. Some people may be born with a genetic predisposition for the disorder, which is then triggered by a combination of factors. Those precipitants may include viral infections; immune system dysfunction; or imbalances in hormones produced in the hypothalamus, pituitary glands or adrenal glands. Complicating the clinical picture is that ME/CFS may not be a single entity, but a spectrum of different diseases that we are currently unable to differentiate.

ME/CFS is surprisingly common. According to an Institute of Medicine (IOM) [report](#) [2] published in

2015, an estimated 836,000 to 2.5 million Americans suffer from ME/CFS, most of whom have not been diagnosed. That could change if the very preliminary report from Stanford University researchers of a diagnostic test is confirmed.

As described in the Stanford [press release](#) [3], the test employs a “nanoelectronic assay,” which measures changes in minuscule amounts of energy as a proxy for the health of immune cells and blood plasma.

*"The diagnostic technology contains thousands of electrodes that create an electrical current, as well as chambers to hold simplified blood samples composed of immune cells and plasma. Inside the chambers, the immune cells and plasma interfere with the current, changing its flow from one end to another. The change in electrical activity is directly correlated with the health of the sample.*

*The idea is to stress the samples from both healthy and ill patients using salt, and then compare how each sample affects the flow of the electrical current. Changes in the current indicate changes in the cell: the bigger the change in current, the bigger the change on a cellular level. A big change is...a sign that the cells and plasma are flailing under stress and incapable of processing it properly. All of the blood samples from ME/CFS patients created a clear spike in the test, whereas those from healthy controls returned data that was on a relatively even keel. The test has been applied to blood samples from 40 people, half of whom have ME/CFS and half of whom who don't. The investigators report that the test clearly identified [all of] those with ME/CFS but was negative in healthy controls."*

These results are very preliminary, and it is important to note that the test measures only a proxy, or surrogate, for what is actually happening in immune cells and blood plasma. The next step should be to investigate the specificity of the test by applying it to patients with a spectrum of diseases, including some whose symptoms overlap with ME/CFS, including fibromyalgia, mononucleosis, and myofascial pain syndrome.

The researchers are also attempting to use the assay to screen for drug-based treatments, by running it before and after adding measured doses of various candidate therapeutic drugs to the patient's blood samples. The theory is that if the blood samples taken from ME/CFS patients still respond poorly to stress and generate a spike in electrical current, then the drug likely didn't work. However, if a drug seems to suppress the spike in electrical activity, that could mean it is having a positive effect on the ability of the immune cells and plasma to “process stress.”

A different approach, by a biopharmaceutical start-up, is based on the thesis that ME/CFS could arise from the up-regulation of a specific receptor (CRF2) in the parts of the brain that govern the sensitivity of the stress response. That would, the theory goes, cause an exaggerated response to a minor stimulus, ultimately leading to the various signs and symptoms, described above, that are commonly observed in ME/CFS. Although there is no good animal model of ME/CFS,

overstimulating CRF2 in healthy rats induces signs and symptoms consistent with the disease in humans; while down-regulating it, with an experimental, synthetic peptide called CT38, eliminates the ability to elicit these signs and symptoms.

A small, early-stage [clinical trial](#) [1] in which the primary endpoint was an assessment of functional parameters following cardio-pulmonary exercise tests, conducted pre- and post-treatment, has been performed with CT38, but the results have not yet been reported.

Additional parts of the ME/CFS puzzle were provided by researchers at an April [conference](#) [4] sponsored by the National Institutes of Health: new evidence of cardiopulmonary and nervous system abnormalities in patients with ME/CFS, which could be the etiology of their exercise intolerance.

It's early days, but we seem to be closing in on understanding, diagnosing, and maybe even treating one of our species' truly awful afflictions. The glacial pace of progress serves as a reminder that we need to pursue a variety of research approaches and to get expeditious regulatory reviews of treatments once they reach clinical trials.

*Henry I. Miller, a physician, is a Senior Fellow at the Pacific Research Institute. He was a Research Fellow at the National Institutes of Health and the founding director of the Office of Biotechnology at the FDA. Please follow him on Twitter: @henryimiller.*

---

COPYRIGHT © 1978-2016 BY THE AMERICAN COUNCIL ON SCIENCE AND HEALTH

---

**Source URL:** <https://www.acsh.org/news/2019/09/16/chronic-fatigue-syndrome-progress-toward-diagnosis-and-treatment-finally-14285>

**Links**

[1] <https://clinicaltrials.gov/ct2/show/NCT03613129>

[2] <https://www.nap.edu/read/19012/chapter/1>

[3] <http://med.stanford.edu/news/all-news/2019/04/biomarker-for-chronic-fatigue-syndrome-identified.html>

[4] <http://palladianpartners.cvent.com/events/accelerating-research-on-myalgic-encephalomyelitis-chronic-fatigue-syndrome-me-cfs-meeting/event-summary-2e5f90defaa4406e8fce234835e11fdf.aspx>