ME/CFS Research Published 21st – 27th August 2020

This week, 1 new research study has been published from the London School of Hygiene & Tropical Medicine. The researchers have proposed a framework for understanding and interpreting the pathophysiology of ME/CFS, which may support future research design and health care interventions.

ME/CFS Research references and abstracts

**Nacul L et al.** **(2020)**

[How Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) Progresses: The Natural History of ME/CFS.](https://www.frontiersin.org/articles/10.3389/fneur.2020.00826/full)

*Frontiers in Neurology* [Epub ahead of print].

**Abstract**

We propose a framework for understanding and interpreting the pathophysiology of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) that considers wider determinants of health and long-term temporal variation in pathophysiological features and disease phenotype throughout the natural history of the disease.

As in other chronic diseases, ME/CFS evolves through different stages, from asymptomatic predisposition, progressing to a prodromal stage, and then to symptomatic disease. Disease incidence depends on genetic makeup and environment factors, the exposure to singular or repeated insults, and the nature of the host response.

In people who develop ME/CFS, normal homeostatic processes in response to adverse insults may be replaced by aberrant responses leading to dysfunctional states. Thus, the predominantly neuro-immune manifestations, underlined by a hyper-metabolic state, that characterize early disease, may be followed by various processes leading to multi-systemic abnormalities and related symptoms. This abnormal state and the effects of a range of mediators such as products of oxidative and nitrosamine stress, may lead to progressive cell and metabolic dysfunction culminating in a hypometabolic state with low energy production.

These processes do not seem to happen uniformly; although a spiraling of progressive inter-related and self-sustaining abnormalities may ensue, reversion to states of milder abnormalities is possible if the host is able to restate responses to improve homeostatic equilibrium.

With time variation in disease presentation, no single ME/CFS case description, set of diagnostic criteria, or molecular feature is currently representative of all patients at different disease stages. While acknowledging its limitations due to the incomplete research evidence, we suggest the proposed framework may support future research design and health care interventions for people with ME/CFS.