

Review

Chronic Fatigue Syndrome Diagnosis and Treatment - Part 2



**Part 1 – February 2009
Definition, Prevalence,
Diagnosis**

**Part 2 - March 2009
Etiology (Cause)**

**Part 3 - April 2009
Treatment**

Etiology

The pathogenesis of CFS is uncertain and likely multi-factorial.[11] Conditions that have been proposed to trigger the development of CFS include viral infection, immune disorders, hypothalamic-pituitary adrenal (HPA) axis dysfunction, and toxic exposure.[1]

Viral Infection

CFS is similar to many infections, including Epstein-Barr (EBV), human retroviruses, human herpesvirus 6, enteroviruses, rubella, Candida albicans, bornaviruses, and Mycoplasma.[1] No one virus has been linked to every case of CFS; however, CFS may be the result of post-viral infection from a variety of viruses.[1,10] Many studies have uncovered a flu-like sickness that precedes the onset of CFS.[10] It is believed that a virus may trigger and lead to chronic activation of the immune system and altered cytokine production. [10]

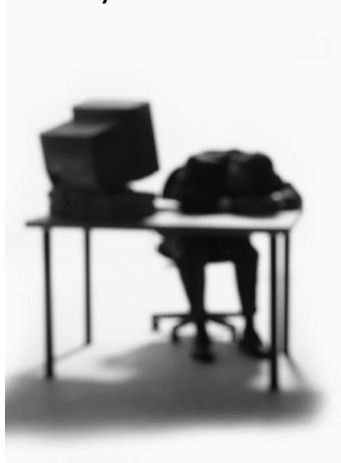
Immunology

“An alteration in cytokine profile, a decreased function of natural killer (NK) cells, presence of autoantibodies and a reduced responses of T cells to mitogens and other specific antigens have been reported” in CFS.[11,12]

Various triggering events such as a traumatic injury or viral infection may lead to the chronic expression of cytokines and then to CFS.[9] Serum chemokine and cytokine profiles have identified a distinct pathogen associated signature for the inflammatory serum chemokines, as well as the pro-inflammatory cytokines.[9] High levels of pro-inflammatory cytokines may explain fatigue and flu-like symptoms.[11]

Studies have reported immune cell phenotype changes and NK cell dysfunction as common manifestations of CFS.[11] Studies have shown that CFS patients are more likely to have a history of co-occurring allergies than are healthy controls, though not all CFS patients have allergies.[1]

There may also be a disorder in the early activation of the immune system involving protein kinase C.[13]





Hypothalamic-Pituitary Adrenal (HPA) Axis

Physical or emotional stress is frequently reported just prior to the onset of CFS.[14] CFS patients produce lower levels of cortisol than do healthy controls which influences the immune system, behavior, and other body systems. [14] While the levels are lower, they are still within the normal range[14].

HPA axis is involved in the adaptive responses to stress.[16] CFS patients report exacerbation of symptoms after physical and psychological stress.[16] One study showed that CFS patients are capable of producing a sufficient cortisol response under some types of stress, but an overall subtle dysregulation of the HPA axis exists.[16]

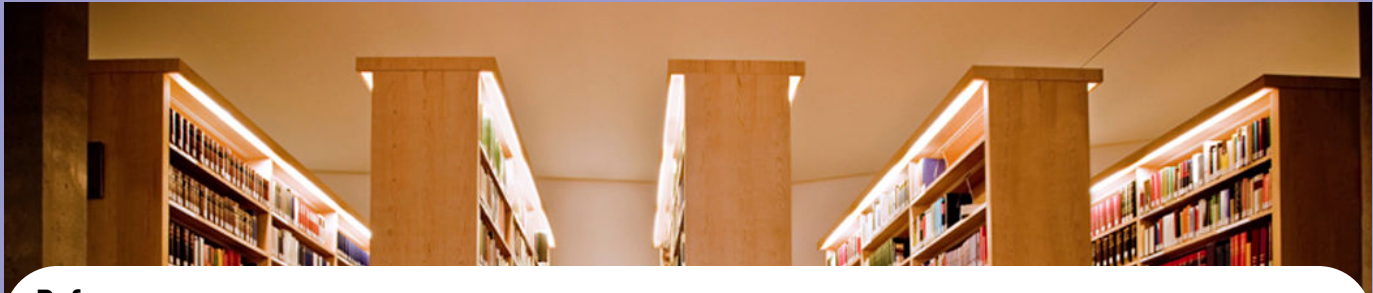
The HPA axis deregulation may exacerbate or perpetuate symptoms.[15] Cortisol acts to suppress inflammation and cellular immune activation. Supplementary cortisol has been shown to temporarily alleviate fatigue.[15]

Neurally Mediated Hypotension

Many patients with chronic fatigue syndrome (CFS) have neurally mediated hypotension when subjected to head-up tilt and disturbances of autonomic regulation of both blood pressure and pulse. [1,17]

New Findings

A study performed by Michael Maes found that the functional symptoms of CFS have a genuine organic cause in the activation of peripheral and central inflammatory and oxidative and nitrosative stress pathways and gut-derived inflammation.[18]



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