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LETTER TO EDITOR

Kynurenine Pathway Hypothesis: The Nature of the Chronic Fatigue Syndrome (CFS) Revisited

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The Nature of the Chronic Fatigue Syndrome (CFS) Revisited

Moderate physicians consider CFS to be missed diagnoses of uncommon illnesses with atypical features. Hartnup (heterozygotes), Lyme and Whipples—like diseases are examples of conditions which fit these clinical ambiguities. The detractors claim it is non-existent. The protractors complain CFS is excluded from standard medical texts. A broad overview of medical literature and support group newsletters, render these opposing views substantially incorrect.

The patient presents with a confounding array of neurological, mental, gastrointestinal, musculoskeletal and perhaps dermatological and visual signs and symptoms. Episodic night sweats can also be reported. Lack of energy, concentration and mobility, limit lifestyle. These symptom constellations evolve and fluctuate in a seemingly random order and can become entrenched. Alcohol intake, protracted steroid therapy and overt or latent infections usually aggravate the course of CFS.

CFS patients often have positive family histories of mixed connective tissue disorders frequently combined with arthralgic and intestinal disorders. In addition, CFS patients may display stigmata of autoimmune inefficiencies with inability to show changes in critical clinical indicators. Temperature, FBE and ESR can be paradoxically "normal".[1] Propensity towards sero-negative variants are reflected in atypical Rheumatoid conditions or B12 deficiency with only achlorhydria and/or neurological deficits as definitive signs.[2] Unusual cytokine profiles are recorded.[3]

The older post Freudian extensions of abstract somatoform theories, limits a clear view of CFS. More recent discovery of malabsorptive and other nutritional neuropathic states[4] make cohesive sense out of the seemingly disparate multisystem CFS patterns. The resolution of diagnostic conundrums are certainly one essential part of the picture. They fail to explain why CFS minorities handle infections and multiple autoimmune disorders in a more malignant fashion, compared with large similar illness cohorts.

Review host defence mechanisms. Starvation, malabsorption, maldigestion and food intolerances are companion to infection. The integrity of interwoven metabolic processes and immune factor formation require adequate substrate levels, an absence of inborn genetic errors and healthy balance of intestinal microorganisms. HIV aids depletes immune competence. Are there comparable organisms which downgrade pre-existing compromised CFS immune systems?

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Tryptophan was the only nutrient supplement to show CFS therapeutic promise.[5] It participates in neuroconduction and immune functions. The Tryptophan/kynurenine pathways with glitched DOI enzyme and gamma-INF and/or other site interactions,[2,6,7] might provide a salient key, to the immunology of chronic smouldering CFS infections[8] and overall morbidity.

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