

CHRONIC FATIGUE SYNDROME as VESTIGE of EARLY AGING and  
NEURODEGENERATION

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Extended Abstract

Chronic Fatigue Syndrome (CFS), the complex clinical symptomatology of chronic illnesses, remains a poorly understood disease with various theories of its elusive etiology and fuzzy pathogenesis. Several possible causation-illustrations factors have been suggested with a particular emphasis on low level radioactive (especially, depleted uranium), and chemical agents, viruses and others, contributing to the broad scope of clinical manifestations. Polarized views of different theory and interest groups maintain CFS as area of sustained controversy, especially in connection with Gulf War and Balkan Syndrome, clearly overlapping with CFS.

Evidence is accumulated that CFS appears to be often induced by a relatively short-term strong or permanent average environmental stress which is followed by chronic pathology, reflected in both neuropsychiatric and metabolic spheres disorder at multiple organism system hierarchy levels.

From both theoretical and experimental standpoint, CFS can be thought as a natural origination model and initiation stage of inducible aging processes involving most of fundamental aspects of its etiology. CFs and starting of inducible aging and neurodegeneration are related to each other by threshold phenomena corresponding to the stages of resistance and exhaustion of stress response with environmental impacts representing progeroid factors which accelerate the aging processes and precipitate the manifestation of CFS as an initial stage to progeroid like systemic disease.

In this connection CFS may be considered as environmentally induced forerunner-vestige of forthcoming accelerated aging and neurodegeneration. CFS etiology and pathophysiology based on the ideas of lossing hierarchy system complexity and disorder of subtle physiological free-radical dynamic quasihomeostasis under extreme or chronic environmental stress encompassing in norm adaptational functional kinetics of O-, N-, and S- centered free radicals and delicate nitric oxide-superoxide-peroxynitrite-SOD-NOS kinetic balance can be regarded as interference of endo- and exogenous variables including interaction of genetic (n-DNA, mt-DNA, and telomeric components), epigenetic, hormonal, and environmental determinants with respective adaptation-compensation-threshold-penalty mechanisms at molecular, cellular, and organisms systems level.

A fundamental target of environmentally and endogenously induced ROS/RNS effects is mitochondrial membrane which is the structural and functional domain of dynamic free-radical redox balance, signal transduction, ion channels, lipid peroxydation, and mtDNA mutations.

In this generalized conceptual framework Chernobyl nuclear accident as an unfortunate unique natural human population experiment and model including various cohorts with different exposure to multiple environmental stress-factors (acute and chronic exogenous and endogenous (radionuclides) irradiation, various chemical agents, including Pb, severe psychological stress, etc) allows to investigate, conceptualize, and illustrate fundamental aspects of CFS etiology and pathophysiology and its triggering parallelism with inducible aging and neurodegeneration processes.

