



## What Do Fibromyalgia, Chronic Fatigue Syndrome, and Dysautonomia Have in Common With Systemic Lupus Erythematosus?

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### Commentary

Complex diseases like systemic lupus erythematosus (SLE) manifest dysregulation of multiple physiological systems. Definable inflammatory and immunologic events in SLE patients often coexist with numerous vague comorbid complaints. The latter encompass features inherent to fibromyalgia (FM), myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), and dysautonomia (DSA), and they seldom wax and wane with successful pharmacologic treatment of SLE itself [1]. Typical symptoms include fatigue, widespread generalized pain, cognitive impairment, non-restorative sleep, irritable bowel syndrome, weakness, palpitations, headaches, dizziness, anxiety, depression, odor and smell hypersensitivity, and medication intolerance.

The spectrum of contributing triggers for immune activation in SLE is quite diverse and includes (but is not limited to) genetic variations, infections, hormonal influences, ultraviolet exposure, stress, medications, faulty apoptosis, vaccinations, racial factors, poor nutrition, micro biota imbalance, and environmental pollution. With regard to environmental pollution, research over the past several decades has primarily focused on chemically-related endocrine receptor disruption caused by organophosphates, heavy metals, polyhalogenated compounds, and phthalates. More recent attention has been directed at chemically-induced alterations of the various epigenetic factors such as DNA methylation, histones, and micro RNAs [2]. The realization that nuclear DNA routinely interacts with mitochondrial DNA has increased the complexity of these investigations. Current epigenotoxicity research, however, does not adequately integrate multiple levels of other environmentally-related physiological abnormalities that far transcend the mechanisms causing immune dysregulation [3]. In FM and ME/CFS alterations in DNA expression are simultaneously accompanied by disturbances of other basic life-sustaining processes, including (but not limited to): (a) many other aspects of mitochondrial function (e.g., the electron transfer system); and (b) matrix macromolecules, enzymes, cytokine production, micro biome diversity, the autonomic nervous system, and the central nervous system [3-7].

Mitochondria are intracellular organelles that retain multiple features of bacterial ancestry. Disturbances of mitochondrial function are far more harmful than simply a reduction in energy production and energy consumption [8,9]. Mitochondria are involved in the regulation of macrophage and T cell activation, and they can foster either a heightened or deficient immune response. The latter can

encompass deficiencies in natural killer cells, down regulation of innate activity, and attenuated B cell receptor expression. Independent of this, inflammatory reactions and activation of innate and adaptive immune responses can be triggered by mitochondrial damage that results in spillage of their contents. Some researchers have implicated neuronal mitochondrial morbidity as the initiating process in multiple sclerosis, whereby autoantibodies may merely represent a reaction to nerve damage rather than the cause (i.e., the antibodies are epiphenomena) [8]. Similar claims have been entertained in autoimmune retinopathy where the presence of anti-retinal antibodies conflict with the absence of pathological inflammation in deteriorating retinal components [6].

Current research investigating chemical toxicity exhibits considerable mechanistic shortcomings for mitochondrial and other diverse physiological malfunctions mentioned above. What other pervasive environmental contaminants can produce such phenomena? Answer: organosiloxane (organosilicon) products. Over the past eighty years more than 60,000 organosiloxane compounds have been synthesized, and they now contaminate every environmental compartment [10,11]. They are pervasive in all aspects of everyday life, and they routinely enter the body via inhalation, dermal absorption, and ingestion [6,12]. These molecules all contain artificial, man-made silicon-carbon bonds, with such bonds never occurring in nature [13]. Organosiloxane molecules, and their degradation products (e.g., silanols and silicic acid), are a "mission impossible" for living organisms to contend with [14]. They can bio integrate into matrix macromolecules causing disruptions in an endless number of overlapping functions [7]. They are injurious to bacteria, disrupt enzyme functions, can cross the blood brain barrier and chelate neurotransmitters such as dopamine, block the parasympathetic activity of acetylcholine, stunt the demand for augmented energy production during exercise, perturb mitochondrial-induced immune activation, can donate methyl groups in vivo to the framework of DNA and to mercury (producing methyl mercury), and enhance nociception [6,14]. And because silicon behaves like a metal at times, the bio integration of organosiloxanes and/or their degradation products into life-sustaining epigenetic molecules can result in alterations of their electromagnetic fields, thereby disrupting communication circuits [6,15,16]. This has relevance not only for human DNA expression and the regulatory networks of human micro RNAs, but can also adversely affect latent viral genomic material that one acquires during his/her lifetime. Indeed, this latter phenomenon has been demonstrated to be relevant in the reactivation process of Epstein Barr virus in the human host. This, in turn, can create the illusion that ME/CFS, FM, and even SLE itself are triggered by an acute viral infection, when in fact the onset of these conditions could be independently initiated by exposure to organosiloxanes (with viral reactivation simply providing an amplification loop). Many of these physiologic malfunctions have been

reported to be transmissible during cell division, thereby explaining the term “chronic” in ME/CFS.

From the above discussion one can readily appreciate that the all-too-common practice of invoking autoimmunity to routinely explain the occurrence of vague phenomena in SLE is a gross oversimplification of what is clearly a much more complicated process. Particularly onerous is the assumption that cognitive impairment in SLE is only due to autoimmune events. Even the decimation of honeybees, which was previously thought to be related to pesticide-induced immune suppression, has now been shown to be caused by organosiloxane surfactants that are added to enhance insecticide stickiness [17]. The resulting distortion of electromagnetic fields in the brains of honeybees prevents them from honing back to the hive, whereupon they fly around in disarray before succumbing to exhaustion. Do you know any patients with ME/CFS who drive into the wrong neighborhood on their way home? And I wonder if honeybees develop dry eyes and dry mouth, because the receptor for acetylcholine in human salivary and lacrimal tissues is a proteoglycan that can readily be blocked by the bio integration of organosiloxane degradation products into matrix macromolecules[7,14]. The “disconnect” of comorbid complaints in SLE patients is not likely to be a “disconnect” at all, and researchers studying vague syndromes need to reassess etiologies and pathophysiologies. Stated another way, understanding tissue injury and disease evolution in SLE extends far beyond the integration of immune dysregulation with chemically-induced mitochondrial dysfunction. This is particularly important in light of the following fact: decades of assertions by physical chemists that organosiloxanes are chemically and biologically inert are now known to be completely untenable. Discerning scientists have always known that the complexity of nature far transcends man’s ingenuity. The real problem in SLE is trying to ascertain what comes first, autoimmunity or environmental toxicity. Or are they inextricably linked to one another whereby one begets the other and vice versa? It’s the old story of the chicken and the egg.

## Conflict of Interest

The author(s) report no conflict of interest in the preparation of this manuscript, and there has been no financial support from any commercial entity for its contents.

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