Review Article

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Do Long COVID Phenomena and Chronic COVID-19 Vaccination-Induced Disorders Share Similar Etiology?

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Clinical features manifested by patients suffering from long COVID following resolution of acute SARS-CoV-2 infections are strikingly similar to chronic ailments caused by COVID-19 vaccinations [1,2]. With regard to COVID-19 vaccine-induced chronic disorders, widespread biochemical disruptions have been postulated to be the etiologic factors driving both disease initiation and disease perpetuation [3]. The evolution and facilitation of these biochemical disruptions are theoretically dependent on a "perfect storm" scenario, whose diverse participants include (but are not limited to): (a) hidden chemical vaccine ingredients; (b) inherent deficiencies of several cytochrome P450 enzyme activities against xenobiotics; (c) innocuous innate channelopathies; (d) altered regulatory T cell function; and (e) malfunction of mitochondrial quantum tunneling, with or without DAMPS (damage associated molecular patterns). The required convergence of these participants may then foster the production of multiple autoantibodies, which, in turn, can create secondary amplification loops that circuitously perpetuate the initial acute adverse vaccine reactions and render them chronic. Multiple reported targets for these autoantibodies include (but are not limited to): (a) adrenergic G protein coupled receptors: (b) channel proteins regulating ion flow in and out of neuronal membranes (e.g., in dorsal root ganglia, or in the brain); (c) neuromuscular junction receptors for acetylcholine; (d) matrix macromolecule components of sensory nerve receptors (e.g., heparan sulfate); (e) matrix macromolecules that bind the preformed mediators of inflammation in mast cells (e.g., chondroitin sulfate); (f) BDNF (heparan sulfate is the attachment site for the serine esterase enzyme that cleaves BDNF to produce an active molecule); and (g) GAD 65 (the enzyme responsible for the production of GABA) [1,3].

Several biochemical disturbances in the prefrontal cortex of long COVID patients exhibiting cognitive dysfunction have recently been identified [4]. These revelations have prompted therapeutic trials of guanfacine (an alpha2A adrenoceptor agonist that inhibits potassium channel signaling to enhance neuronal synapses) and N-acetylcysteine (an anti-oxidant that protects mitochondria and reduces microglia-derived neuroinflammation). Cognitive improvement following their combined use has been demonstrated [4]. Two questions come to mind from these observations: (a) are chemical toxicities the initiators of such disturbances, and if so, where would they come from; and (b) are there cytochrome P450 xenobiotic enzyme deficiencies in long haulers?

Chronic SARS-CoV-2 persistence in the GI tract following resolution of its acute infectious process has been repetitively identified [5]. Intestinal epithelial lining cells exhibit ACE-2 on their surface, which is the receptor for the spike protein of SARS-CoV-2. This interaction may lead to some enhancement of toxin absorption into the body, but this absorption does not necessarily have to include enterotoxins. Intestinal bacteria have the capacity to demonstrate geomicrobiology, whereby the dietary ingestion of chemicals attached to daily food products prompts catabolism followed by anabolism [6,7]. Stated more simply, these bacteria

have the ability to break down organosiloxanes and other chemicals (e.g., pesticides, insecticides, polyhalogenated hydrocarbons), and then utilize the carbon fragments for the synthesis of new diverse chemical species. The identification of these new species and their potential adverse effects on the body have not been properly studied. Assuming that one has a normal complement of cytochrome P450 enzymes in the liver, these absorbed toxins may have little or no consequences. But if an individual exhibits one or more genetic defects causing deficiencies in enzymes that metabolize xenobiotics, biochemical chaos could ensue. Several such enzyme reductions have been demonstrated in individuals suffering from chronic rheumatic and neurologic ailments initiated by hidden toxic chemicals in the Gardasil vaccine [8]. Biochemical disruptions related to cytochrome P450 enzyme deficiencies have also been postulated to causally participate in the production of similar ailments following COVID-19 vaccinations, because COVID-19 vaccines also contain a large variety of toxic chemicals [1,3]. In unpublished data, defects in the CYP3A4 gene and the CYP2C19 gene have been identified in long COVID individuals manifesting cognitive dysfunction. Both of these gene's code for enzymes active against xenobiotics. Unpublished data has also demonstrated clinical improvement in the cognitive dysfunction of long COVID patients when xifaxin was prescribed. Xifaxin has been shown to increase the function of the CYP3A4 gene.

The diversity of unresolved physical and psychological symptoms comprising long COVID have been extensively reviewed [9]. Despite this, causation mechanisms and definitions of the various subtypes remain unclear. However, clinical abnormalities and physiological derangements in long COVID are remarkably similar to the chronic ailments manifested by some recipients of various COVID-19 vaccinations. This implies that widespread biochemical disturbances may play a key role in initiating and perpetuating both conditions. The potential existence of faulty xenobiotic metabolism in these two entities is worthy of further investigation.

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