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Vaccination Induced Diseases and their Relationship to Neurologic Fatiguing Syndromes, Channelopathies, Breast Implant Illness, and Autoimmunity *via* Molecular Mimicry

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Abstract

Current theories utilized to explain vaccination induced diseases center mainly around autoimmune phenomena mediated by molecular mimicry to viral or bacterial antigens, bystander cellular activation, or the presence of adjuvants and hidden toxins in vaccine ingredients. Proponents of these disorders have encountered intense controversial feedback from other vaccine researchers who point out the acknowledged infrequency of vaccine complications, overly simplistic and illogical ideas of aberrant immune activation, disease heterogeneity, vaccine efficacy, and the failure to appreciate benign observations inherent to similar environmental exposures. In this report novel and plausible alternative mechanisms of disease causation are proposed by comparing the initiation and perpetuation of vaccination induced autoimmunity with biochemical and physiological disturbances occurring in seemingly disparate disorders, namely neurologic fatiguing syndromes, breast implant illness, and channelopathies. Such disturbances are capable of producing multiple overlapping amplification loops of immune dysfunction mediated by the deleterious comingling of complex interactions between indigenous host factors, mitochondria, immunocompetent cells and toxic vaccine ingredients, all of which then circuitously reinforce each other in a sustained manner.

Keywords: Vaccinations; Immunizations, Autoimmunity; Chronic fatigue syndrome; Fibromyalgia; Dysautonomia; Small fiber neuropathy; Channelopathies; Breast implants; Silicone; Organosiloxanes; Rheumatoid arthritis

Introduction

Vaccination induced diseases encompass a diverse group of mostly (but not exclusively) autoimmune and autoinflammatory disorders that typically appear as case reports in medical journals. These reports encompass acute and chronic systemic ailments and other adverse immunization experiences encountered by practitioners in multiple disciplines, including rheumatology, neurology, hematology, immunology, endocrinology, ophthalmology, otolaryngology, and family medicine [1-5]. Although the biological essence of vaccines is to provide resistance and/or protection against dangerous microbes, and although there is abundant evidence of their overwhelming efficacy and safety, this does not detract from the occasional occurrence of serious side effects or even death. The challenge, therefore, is to determine the population at risk for adverse events by considering novel areas of investigation.

Neurologic Fatiguing Syndromes and Breast Implant Illness

Neurologic fatiguing syndromes encompass a variety of diverse ailments that sixty years ago were vague and rare but are now vague and common. Typical examples include chronic fatigue syndrome/myalgic encephalomyelitis, fibromyalgia, dysautonomia, small fiber neuropathy, complex regional pain syndrome (also known as reflex sympathetic dystrophy), postural orthostatic tachycardia syndrome, and a sub-category of non-autoimmune vaccination induced disorders. Considerable symptom overlap exists among these entities and includes (but are not limited to) fatigue, chronic widespread pain, headaches, cognitive dysfunction, alterations of gastrointestinal motility, paresthesias, orthostatic fainting, tachycardia, non-restorative sleep, difficulty in concentration, dry eyes, muscle twitching, protracted morning stiffness, anxiety, food intolerances, odor and smell hypersensitivity, unexplained chest pains, central sensitization, muscle weakness, fevers, dizziness, and increased sensitivity to innocuous stimuli.

Recent demonstrations that nano needle abnormalities of cellular impedance can be repetitively demonstrated in patients with CFS/

ME have given credibility to this disorder [6]. Other biochemical and physiological derangements have been unearthed in many of the neurologic fatiguing syndromes, including mitochondrial dysfunction, cytokine elevations, immune dysregulations, altered levels of metabolites, epigenetic disturbances, transcriptome changes, hypothalamic dysfunction, microglia and mast cell activation, altered enzyme activity, & neurotransmitter blockade [7-16].

Multiple publications have suggested linkage of these biochemical and physiological derangements in neurologic fatiguing syndromes to worldwide environmental contamination by 60,000 organosiloxane (silicone) compounds synthesized over the past eighty years [1,17-24]. The basis for these assertions is quite straightforward: organosiloxanes (silicones) are chemicals with artificial silicon-carbon bonds that do not exist in nature, and which are a metabolic mission impossible for any living organism on the planet to cope with. Prior assertions by physical chemists that these compounds are chemically and biologically inert are now known to be completely untenable. Organosiloxanes and/or their degradation molecules have been identified in frogs, seals, routine household inhabitants, and honeybees [25,26]. In addition to their capability to produce the biochemical and physiological derangements mentioned above, these chemicals have the capacity to inhibit mitochondrial-induced cell danger responses that have been mounted against other environmental contaminants [27]. Thus, silicones can act as "the straw that broke the camel's back." The ultimate nasty organosiloxane culprits are silicone gel-filled breast implants, because their voluminous polymer loads cause extreme clinical ailments and biochemical derangements that are (a) similar to the multiple phenomena occurring in those afflicted with neurologic fatiguing syndromes, and (b) are now acknowledged by the FDA to have adversely affected hundreds of thousands of recipients in the USA over the past thirteen years [1,21,28-30]. This comparison is not intended to equate the two disorders, as that would be a gross oversimplification for the toxicity caused by implanted silicone devices. In breast implant illness the chronological pattern of disease evolution (which simulates a dose-response curve), when coupled with various types of skin rashes, pigment changes, recurrent infections, hair loss, photosensitivity, metallic taste and nail changes, are all testimonies to its unique existence despite multiple other overlapping similarities with neurologic fatiguing syndromes [28,31,32]. It should also be noted that breast implant illness exhibits "autoimmune features," but these arise from secondary amplification loops and are not the primary stimuli producing the disease [17,29,31,32]. In essence, breast implant illness has been a huge inadvertent experiment that (as will be outlined below) has direct relevance to both autoimmune and non-autoimmune vaccine induced diseases.

Channelopathies

Rapidly emerging discoveries in channelopathies have added another dimension to this discussion. Sodium, potassium, and other ions routinely fluctuate in and out of cells through pores (channels) in cell membranes. Channelopathies are diseases caused by disturbed function of ion channel components and/or the proteins that regulate them [33]. These diseases are either congenital (i.e., from a mutation in one or more genes encoding the proteins) or acquired. Examples of the latter can occur from autoimmune attack on ion channel proteins, ligand anomalies, or from chemical toxicity. Voltage gated sodium channels are responsible for electrical action potential initiation and propagation in nerve cells and muscle cells. The membrane proteins that form such channels allow for bidirectional flow of the sodium ion. A voltage gated channel is different from a ligand activated channel, and both are different from a mechanically activated channel (e.g. *via* a pressure sensation). The regulated flow of sodium in and out of cell membranes is accompanied by a similar regulated flow of potassium, and both are responsible for restoring the membrane potential after an electrical signal is sent. It is important to note that sodium ions cannot traverse potassium channels, and potassium ions cannot traverse sodium channels. Channels also exist for other ions, such as chloride and calcium. In the case of a gene mutation (e.g. SCN2A), excitation, propagation, and inhibition of electrical nerve and muscle signals are faulty due to disordered regulation of sodium flow through the gated channels. The clinical features of SCN2A mutation are profound and manifest themselves in the first few months of life [33]. Such clinical phenomena include (but are not limited to) myotonia, epilepsy, spasms, intellectual developmental delay, widespread generalized pain, fatigue, autism, hallucinations, cognitive dysfunction, weakness, dyskinesia, and gastrointestinal disturbances. There are more than 400 different genetic variants that alter channel proteins [34]. These cause a broad range of clinical presentations that are not yet fully understood, many of which can be asymptomatic under innocuous conditions unless activated by adverse environmental events (e.g, fever accompanying a viral infection, chemical exposures, vaccinations, physical injuries, or severe emotional upset). Channelopathies do not just affect efferent and autonomic nerve pathways. Pain sensation arises from activation of sensory nerve electrical signals. Such activation is quite complex and is influenced not only by voltage gated, ligand gated and mechanically activated ion channels, but also by glial cells, immunocompetent cells, inflammation, kinases, growth factors and neurotransmitters [34]. Paresthesias, dysesthesias, and localized or widespread chronic pain can develop from a variety of ion channel malfunctions. Ion channels are also present across cell membranes of immunocompetent cells, including (but not limited to) regulatory T cells, neutrophils, lymphocytes, dendritic cells, macrophages, eosinophils, and natural killer cells [35]. Even mitochondrial membranes have ion channels [34]. Proper mitochondrial function is responsible for meeting metabolic demands if functions become maladapted they can initiate neurologic fatiguing syndromes, trigger activation of innate and adaptive immune responses (leading to autoimmune and autoinflammatory disorders), increase susceptibility to other chronic diseases, and accelerate aging [34,36,37]. Thus, channelopathies are capable of creating multiple amplification loops of immune dysregulations that circuitously reinforce one another.

The proper functioning of ion channels are also important for mast cell stabilization and/or degranulation. When mast cells degranulate, their inflammatory mediators can cross the blood brain barrier and activate microglia cells, thereby promoting what has been referred to as neuroinflammation [1,11].

Therefore, a key question to be asked is: can organosiloxanes, by themselves, produce pathologic changes in cell membrane ion permeability? If the answer is yes, then such a channelopathy would be capable of augmenting the biochemical and physiological derangements inherent to organosiloxane-induced neurologic fatiguing syndromes and breast implant illness. Organosiloxanes and their degradation products (e.g., silanols), can adhere to proteins *via* hydrophobic bonding, thereby inducing conformational changes that may translate into altered channel functions [17,28]. Secondly, silicones readily adhere to phospholipids present in the bilipid layer of cell membranes. Thirdly, silicones bind heavy metals, such as platinum and rubidium, which are utilized to promote polymerization of organosiloxanes during their manufacturing process (and these heavy metals do not fall out of the soup mixture at the end).Many proteins incorporate minerals (e.g., zinc) as cofactors for proper functioning. Heavy metals displace these minerals and alter protein function. The myriad of

clinical manifestations caused by heavy metal toxicity have been comprehensively reviewed elsewhere [38]. Fourth, organosiloxanes can biointegrate into matrix macromolecules, including proteoglycan receptors on cell surfaces, causing disruptions in an endless number of overlapping functions [17]. Lastly, organosiloxane degradation molecules in the form of silanols can easily produce epigenetic changes *via* enhanced DNA methylation [1,21,23,25]. This, in turn, may result in altered gene expression and the production of autoantibodies. As an example, researchers have reported positive antinuclear antibodies in one-third of ailing breast implant recipients. A relevant extension of this observation could be organosiloxane induction of autoantibodies directed against channel proteins, thereby altering ion exchange. If a subclinical channelopathy is simultaneously present, the autoimmune effect could be enhanced.

Novel Mechanisms for Vaccination Induced Diseases

From the above discussion one can readily appreciate that organosiloxane-induced alterations of cell membrane ion permeability are plausible occurrences that can be mediated by autoimmune and non-autoimmune mechanisms. Organosiloxanes (silicones) and silica (silicon dioxide) are known hidden ingredients in at least sixteen commonly administered vaccines [1,2]. Vaccination induced autoantibodies directed against potassium channel proteins have been reported to cause permanent alterations of normal muscle function [39], and autoimmune induced alterations in ion channel function have been reported in patients suffering from repetitive, and even life-threatening, cardiac arrythmias [40]. Applying these observations to the abundant case reports of vaccination-induced rheumatologic and neurologic diseases creates an entirely new perspective regarding the long-standing controversy encompassing the validity of such diseases [2,3,5]. There are numerous reasons for such controversy, including (but not limited to): (a) the lack of randomized, double-blind placebo controlled studies; (b) any single autoimmune disorder has been observed to arise following multiple different immunizations; (c) single immunizations have been implicated as the cause of multiple autoimmune and/or autoinflammatory conditions; (d) in a timely manner regulatory T cells routinely dampen down the normal simultaneous production of autoantibodies following immunization, thereby casting considerable doubt on the main theory of molecular mimicry; (e) time intervals extending from the day of vaccination to the appearance of disease symptoms and signs are highly variable, ranging from 24 hours to four weeks; and (f) no one has as yet defined the population at risk for such infrequent occurrences [2-5]. In addition, some immunologists routinely dispute all theories of vaccine-induced autoimmunity by directing attention to the "accepted and well delineated" chronological evolution of immunocompetent cell activation inherent to the initiation and progression of diseases like rheumatoid arthritis. These immunologists give little credence to the idea that rheumatoid arthritis may be a syndrome rather than a disease, and that its varied initiation by physical trauma, sustained emotional lability, insecticides, and multiple other triggers bears little or no relevance to "classical" concepts [41,42].

Perhaps the initiation of autoimmune diseases by vaccinations is a three or four step process rather than a single event. The necessary first step would be the presence of indigenous host factors capable of inhibiting regulatory T cells from eliminating autoantibodies that accompany routine immunizations. Subclinical channelopathies could represent such host factors, and their innumerable subtypes could account for disease heterogeneity by virtue of which cells, tissues and organs share the same anomaly. The second step, in concert with organosiloxane induced epigenetic disturbances already outlined, would be the simultaneous "activation" of the subclinical channelopathy in regulatory T cells by parenterally administered toxic vaccine ingredients (which includes organosioxanes). The ensuing dysfunction of ion channel protein activity in regulatory T cells could be immediate or delayed, but in either case there would be dysfunction of these T cells themselves. This, in turn, could allow incidental vaccine induced autoantibodies to persist indefinitely. The third step could encompass several scenarios. On one hand these autoantibodies may be directed at specific tissues and organs culminating in a specific autoimmune disease. On the other hand these autoantibodies may be directed at ion channel proteins in other immunocompetent cells and mitochondria (all of which share the same subclinical channelopathy present in the regulatory T cells, and all of which also share the same simultaneous exposure to the parenterally administered toxic vaccine ingredients). These processes are not likely to be mutually exclusive. Indeed, complex chronological scenarios and complex disease identification could be the rule rather than the exception. One also needs to consider that ion channel dysfunction of multiple immune competent cells and mitochondria might already be underway (as a fourth step) even before autoantibodies went into "attack mode." These hypotheses can explain why vaccination induced autoimmune and autoinflammatory diseases are infrequent, because such occurrences would depend on a "double or triple or quadruple hit."They would also explain why the population at risk for such occurrences is not dependent on a familial autoimmune diathesis. There is also another factor to consider, namely that the most recent immunizations administered (singly or in combination) may not necessarily be the sole culprits implicated in disease causation. The process of vaccination induced disease may sometimes be dependent on cumulative immunization exposures.

It remains to be seen if removal and/or replacement of hidden toxic components of vaccines will avert and/or lessen autoimmune complications. It should also be noted that not all vaccination induced diseases are primarily autoimmune in nature, as some immunization disorders exhibit overlapping clinical phenomena reminiscent of each and every neurologic fatiguing syndrome [1]. In these scenarios there are "autoimmune features," but just like neurologic fatiguing syndromes and breast implant illness these features arise from secondary amplification loops and are not the primary stimuli producing disease symptoms [1,17-24,28,31,37].

Conclusion

In summary, the development of neurologic fatiguing syndromes, breast implant illness and vaccination induced disorders are capable of being initiated by a myriad of organosiloxane-induced biochemical and physiological disruptions, all of which can then be augmented by organosiloxane-induced alterations in cell membrane ion channel permeability. Differences in severity among patients already afflicted with neurologic fatiguing syndromes and/or breast implant illness and/or vaccination induced disorders may, in part, be related to the theoretical presence in some individuals of subtle genetic coding mutations for channel proteins, which then become clinically relevant under conditions of organosiloxane exposure. The circuitous amplification loops of immune dysregulations occurring in individuals suffering from neurologic fatiguing syndromes and breast implant illness are to be expected, but they should be relegated in importance to secondary exacerbation phenomena and not primary events. The plausible circuitous amplification loops of immune dysregulations occurring in individuals suffering from vaccination induced disorders are likely to be much more complicated and will require sophisticated research protocols to sort them out.

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