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Why are Vaccination induced Rheumatologic Disorders so Diverse?

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The past three decades have witnessed an expanding list of case reports claiming initiation of multiple rheumatic diseases by vaccinations [1-7]. Examples of these disorders include rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), polymyalgia rheumatica (PMR), giant-cell arteritis (GCA), reactive arthritis (ReA), dermatomyositis (DM), systemic juvenile idiopathic arthritis (sJIA), necrotizing vasculitis (NV), "adult-like" JIA, and neurologic fatiguing syndromes (NFS). Equally diverse are the immunizations associated with triggering these ailments, including (but not limited to) vaccines protecting against diptheria, tetanus, pertussis, influenza, varicella, MMR (measles, mumps, rubella), hepatitis B, human papillomavirus, and hemophilus influenza B.

Potential mechanisms of disease causation in these scenarios have typically focused on autoimmune reactions triggered by vaccinations. Similar claims have been made for other vaccine-induced case reports, including Guillain Barre syndrome, hemolytic anemia, thrombocytopenia, optic neuritis, sensorineural hearing loss, chronic demyelinating conditions, neuromyotonia, and transverse myelitis [8]. Molecular mimicry has received the most attention of all the various immunologic theories because it seems apparent that similarity exists between some vaccine-containing antigens and self-antigens in the body [9]. This observation is then postulated to be the stimulus for the production of cross reacting autoantibodies capable of initiating and sustaining tissue damage. However, vaccine-induced autoantibody production appears to be surprisingly universal and is a transient phenomenon of no clinical consequence in apparently healthy people [10]. This observation suggests that host factors precipitating regulatory T cell dysfunction after immunization are primarily responsible for the occurrence of chronic autoimmune reactions. The acknowledged infrequency of these aberrant vaccine-related disease states does not detract from the urgency of trying to determine the population at risk. Equally confusing are the observations that a single vaccine may be capable of initiating a wide variety of different autoimmune disorders, and that any single disorder may be triggered by a wide variety of different vaccines. Over the past decade a new theory known as ASIA (autoinflammatory syndrome induced by adjuvants, or Shoenfeld's syndrome) attempted to offer novel insight into the discussion, but this syndrome (and the animal models it has relied upon) have recently been refuted and/or subjected to significant controversy [11-13].

Little to no attention has been directed towards other potential mechanisms of vaccine-induced illness, namely: hidden toxicity exposures, channelopathies, disturbances in mitochondrial function, alterations of matrix macromolecules, heavy metal presence, mast cell dysfunction, and autoinflammatory reactions (i.e., reactions initiated by the innate arm of the immune system, as opposed to adaptive (lymphocyte mediated) immune responses). Each of these has the capacity to explain many of the disease states observed in ailing vaccine recipients. They also have the capacity to circuitously reinforce one another and simultaneously create multiple overlapping amplification loops of autoimmune phenomena. Table one lists vaccines that contain hidden toxicity in one or more components: (a) Polysorbate-80 (PS-80); (b) an immunostimulatory compound (ISCOM); and (c) sodium dihydrogen phosphate dihydrate (SDPD). A recent publication has linked these three vaccine ingredients to chronic disorders exhibiting features of multiple neurologic fatiguing syndromes [6]. Directly or indirectly these three ingredients contain organosiloxanes (silicones) and/or silica (silicon dioxide). Silica exposure has a long and sordid history of human toxicity. Silicones, in the form of gel-filled breast implants, are now acknowledged to be the cause of a genuinely novel systemic illness orchestrated by dozens of biochemical disruptions in the body [14,15]. These devices have

recently precipitated a recurrent public health debacle in more than 350,000 women in the USA, and the Food and Drug Administration (FDA) now requests black box warnings and other illness-related information that surgeons must provide prospective recipients prior to anticipated insertion. Autoimmune vaccine relevance occurs when the parenterally administered organosiloxanes and/or their degradation molecules (e.g., silanols) adversely affect epigenetic factors (e.g., DNA methylation), resulting in altered DNA expression and the production of autoantibodies [16]. As an example, researchers have reported positive antinuclear antibodies (ANA) in one-third of ailing breast implant recipients. The hidden toxicity exposure to organosiloxanes in vaccines can also adversely affect the functions of matrix macromolecules and the pre-formed mediators of inflammation contained in mast cells, both of which have been reviewed elsewhere [6,17]. The former can cause abnormal lymphocyte homing and block neurotransmitter receptors; the latter can precipitate neuroinflammation. The manufacturing process of organosiloxanes utilizes heavy metals, and these do not fall out of the soup mixture at the end [14]. Heavy metal toxicity precipitates a wide variety of clinical ailments and has been extensively reviewed elsewhere [18].

Vaccines Containing Polysorbate-80 (PS-80)
DTaP & TDaP (diptheria, tetanus, and acellular pertussis)
Rotavirus (Rotateq)
PCV 13 (pneumococcal conjugate vaccine)
MMR (measles, mumps, rubella)
Hepatitis A
Influenza
Meningococcal
Vaccines Containing an ISCOM (Immunostimulatory Compound)
Hepatitis B
HIV (human immunodeficiency virus)
Influenza over 65 years of age
Shingrix (for herpes zoster, or shingles)
Malaria
Gardasil (for HPV, or human papillomavirus)
Cervarix (for HPV)
Vaccines Containing Sodium Dihydrogen Phosphate Dihydrate (SDPD)
Hepatitis B
Vaccines with None of the Above
IPV (inactivated polio vaccine)
HiB (Hemophilus influenza B)
Varivax (chicken pox)

Sodium, potassium, calcium, and other ions routinely fluctuate in and out of cells through pores (channels) in cell membranes [19]. Channelopathies are diseases caused by disturbed function of ion channel components and/or the proteins that regulate them. These disorders 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 [20], protein malfunction from organosiloxane presence [21], and chemical toxicity from air pollution [22]. Vaccination induced autoantibodies directed against potassium channel proteins in neurons have been reported to cause chronic and permanent alterations of normal muscle activity and seizures [23]. Ion channels are also present across cell membranes of all immunocompetent cells, including lymphocytes, T regulatory cells, neutrophils, macrophages, dendritic cells, natural killer cells, mast cells, eosinophils, and basophils [24]. Since both adaptive and innate immune dysfunction are indigenous to numerous rheumatic disorders, research is clearly needed to determine if vaccine-induced autoantibodies can readily develop against channel proteins in these cells. If eventually proven, then additional evidence for vaccine induced initiation of autoimmune and autoinflammatory phenomena will exist. The same potential vaccine-induced immune disturbances exist for mitochondria, because ion channels are also present across mitochondrial membranes [25]. Proper mitochondrial function is responsible for meeting metabolic demands - dysfunction and/or spillage of mitochondrial organelles are capable of triggering chronic inflammatory and autoimmune diseases, neurologic fatiguing syndromes, and even accelerated aging [26,27].

From the above discussion one can readily appreciate that the development of both autoantibody and chemically induced channelopathies following vaccination are not only plausible but are also capable of creating multiple amplification loops of immune dysfunction that circuitously reinforce one another. Once pathologically activated, the immune system itself can induce other channelopathies that adversely affect neurological, cardiovascular and endocrinologic systems, adding considerable complexity to the process. When coupled with all the other mechanisms of vaccine induced disturbances described in this commentary, it is no surprise that there exists an ever-expanding list of publications reporting on the heterogeneity of autoimmune, autoinflammatory, and neurologic fatiguing syndrome disorders triggered by immunizations.

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