

Covid-19 Vaccine-Induced Autoimmune Disease: Case Study with Therapeutic and Dietary Regimen

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Abstract

The incidence rate of vaccine induced autoimmune disease is unknown, but frequently reported. Here I report my own mRNA vaccine induced autoimmune disease that has been mitigated by a diet strategy, and reference several other cases, including a life threatening episode treated with a polypharmacy approach. I discuss the mechanisms by which a whole food plant based diet may mitigate autoimmunity, including the mitigation of autoantibodies that target neural tissue and likely cause many of the symptoms.

Introduction

Whether one has been infected with SARS-CoV-2 or had the Covid-19 vaccine, a robust innate and adaptive immune response is elicited. Recent studies provide evidence that the adaptive immune response can persist at high levels for over 6 months after vaccination as measured by sera antibody titers (Doria-Rose et al, 2021). Sera antibody titers against the SARS-CoV-2 virus are significantly higher in the vaccinated compared to those who are infected (Assis et al, 2021; Bartsch et al, 2021). For an unknown number of people, either of these events may lead to autoimmune disease (Guimarães et al, 2015); here called SARS-CoV-2 induced autoimmune disease (Ehrenfeld et al, 2020), and Covid-19 vaccine induced autoimmune disease (Toussirost and Bereau, 2015; Segal and Shoenfeld, 2018). While the two conditions may share similar mechanisms of an impaired, hyper immune response, long Covid may have additional mechanisms such as viral persistence (Wang et al 2020; Neurath

et al, 2021). Viral persistence, even at low levels, can lead to a number of consequences, including the release of miRNA packaged into exosomes that induces a pro-inflammatory, Warburg-like effect in surrounding cells (Yoshikawa et al, 2019; Proal and VanElzakker, 2021). Common to both conditions, i.e vaccine induced and Covid-19 induced autoimmunity, susceptibility to infection or severe outcomes may include the effects of previous infections or vaccinations. For example, superantigen-mediated T cell activation can trigger broad B cell activation, and production of autoantibodies against a range of tissues has been shown in multi-inflammatory syndrome (Consiglio et al, 2020), and in patients with acute COVID-19. The spike protein, whether a part of the virion or of the Covid-19 vaccine, contains a superantigenic motif known to elicit a hyperinflammatory adaptive immune response (Cheng et al, 2020). Also, chronic activation of the immune system by viral persistence can induce autoimmune responses, and molecular mimicry between components of a pathogen and host tissue can lead to specific post-infectious autoimmunity. Structural similarity between human neuronal antigens and SARS-CoV-2 proteins exists. A particular form of autoimmunity described in long COVID is postural orthostatic tachycardia syndrome, a form of autonomic dysregulation that is possibly induced by functional autoantibodies that target G protein–coupled receptors on neurons (Brodin et al, 2022). Another type of autoimmunity relevant to SARS-CoV-2 infection is the production of neutralizing autoantibodies to type I interferons, explaining a sizeable fraction of cases of hypoxemic COVID-19 pneumonia (Bastard et al, 2021). If such neutralizing autoantibodies are present before SARS-CoV-2 infection, due to prior infections or vaccinations, then a patient is clearly at risk of developing severe acute COVID-19 or vaccine induced autoimmune disease. Neutralizing autoantibodies may also appear after SARS-CoV-2 infection, in which case they might instead enable viral persistence, the formation of a viral reservoir and long COVID.

Maguire (2022) has suggested a means to develop vaccines that better prevents the spread of virions and also reduce the probability of vaccine injury. However, current Covid-19 vaccine induced autoimmune disease can be severe in adults (de Bruijn et al, 2021; Kaulen et al, 2022) and children (Buckhorn et al, 2021), leading to hospitalization.

Life threatening autoimmune disease from Covid-19 vaccination has been successfully treated with a combination of 1. plasma exchange to clear autoantibodies from the blood, 2. Corticosteroids to reduce inflammation, 3. Rituximab to deplete beta cells, and 4. Caplacizumab an anti-von Willebrand factor to clear blood clots (de Bruijn et al, 2021).

Autoimmune encephalitides ((Zlotnik et al, 2021), venous sinus thrombosis (Finisterer and Nics, 2021; Sharifian-Dorche et al, 2021), intracranial hemorrhage with venous sinus thrombosis occurring in the same patient (Purkayastha et al, 2021), and glial fibrillary acidic protein astrocytopathy (GFAP-A) can result following the second dose of an mRNA vaccine (Koh et al, 2022). Autoimmune encephalitis is difficult to diagnose with current clinical diagnostics and therefore often goes untreated (Graus et al, 2016). Autoantibodies can persist for at least 6 months following even mild Covid-19 disease (Liu et al, 2021; Su et al, 2022). Neurological symptoms will result (Patone et al, 2021; Finisterer, 2022), including memory and attention deficits for up to 9 months (Zhao et al, 2022), and brain autoimmunity with attack of myelin in neurons may result (Gupta and Weaver, 2021; Shabani, 2021). Autoantibodies acting on vascular endothelial cells (Bouillet et al, 2013) in the part of the blood supply that feeds the brain, can cause thrombotic thrombocytopenia (Zuo et al, 2020; Gunther et al, 2021), and cerebral venous sinus thrombosis (Finsterer, 2021), and may also underlie the generalized report of “brain fog” in such patients and other forms of encephalopathy (Huang and Huang, 2022). Recent studies have found that spike proteins in SARS-CoV-2 attach to vimentin (Amraei et al, 2022), which is present at the extracellular surface of endothelial cells. This is a possible mechanism underlying the vaccine induced vascular abnormalities. Autoantibodies are also known to attack neutrophils, a key component of the innate immune response, and therefore could be an important reason for severe Covid-19 in those with autoimmune disease, potentially even that induced from vaccination (Weiner and Segelmark, 2016). Douaud et al (2022) found that Covid-19 whether severe or non-severe (not hospitalized), have significant brain damage. Whether this brain damage is the result of a hyperimmune response is not known, and therefore whether a hyperimmune response to vaccination could be resulting in the

same brain damage is not known. However, because many of those autopsied after Covid-19 with CNS symptoms have not been found to have virions in their brain tissue, but do have immune cells present, a hyperimmune response is likely causative (Matschke et al, 2020). Concerning too is that mRNA vaccines may introduce DNA into the host genome, thus potentially introducing viral proteins to the host immune system for extended periods. Unlike what physicians in the media have said (Orfit, 2021), humans possess robust reverse transcriptase enzymes that can write RNA sequences into DNA (Chandramouly et al, 2021), and the possibility exists that mRNA vaccines may introduce a DNA message into human genomes (Zhang et al, 2021; Alden et al, 2022). More work is required to provide good evidence whether this is happening in vaccinated humans (Doerfler, 2021).

As Danice Hertz, MD has written in a response to an article about Long Covid, "There are many thousands of people who have suffered a similar neurological syndrome as a result of receiving a Covid vaccine. I am one of those people and have severe neuropathic pain from head to toe as well as tinnitus, dizziness, imbalance, blurred vision, fatigue, headaches for 14 months now. Many of us have been diagnosed with small fiber neuropathy, dysautonomia and mast cell activation syndrome. It is time that these vaccine reactions be acknowledged, and that research be conducted to help understand the mechanism of injury so better treatments can be available to help those like me who have suffered terrible injury from the vaccines" (George, 2022). Recently, Gregory Poland, MD, director of the Mayo Clinic's Vaccine Research Group in Rochester, Minnesota, reported his severe tinnitus after receiving the second dose of a mRNA covid-19 vaccine. "It was like someone suddenly blew a dog whistle in my ear," Poland told MedPage Today. "It has been pretty much unrelenting." Since then, Poland said he has been experiencing what he describes as life-altering tinnitus. Commenting on his symptoms, he "can only begin to estimate the number of times I just want to scream because I can't get rid of the noise or how many hours of sleep I've lost," (Henderson, 2022).

In this analysis and case study of vaccine induced autoimmune disease, I'll first assume that the vaccine was manufactured correctly, and transported and stored appropriately so that the vaccine itself was normal. This, of course, is a big assumption given recent reports of manufacturing irregularities at the vaccine manufacturing facilities (Kansteiner, 2021). Because the VAERS vaccine adverse event reporting system in the US is voluntary, and few physicians or individuals who have been vaccinated report their injuries (Vaccines and The National Vaccine Injury Compensation Program, <https://dash.harvard.edu/handle/1/9453695>), the rate of vaccine induced injury is unknown. "The concern of some physicians about potential legal liability for an adverse event following vaccination was cited by some participants as a reason for underreporting in VAERS" (IOM, 1997). According to Ross et al (2010), "less than 0.3% of all adverse drug events and 1-13% of serious events are reported to the Food and Drug Administration (FDA). Likewise, fewer than 1% of vaccine adverse events are reported" (Ross et al, 2010). The underreporting of AEs occurs for many different vaccines that are routinely used (Cunningham, 2010). The problem of underreported AEs is significant (about 85%) for all drugs, not just vaccines (Hazell and Shakir, 2006). Some studies have attempted to capture the true rate, such as that for vaccine induced myocarditis (Barda et al, 2021), but these studies are rare and are usually poorly executed, if only because the data base from which the adverse events are taken is of poor quality. Here's part of the problem too: if one has been injured by the vaccine, symptomatic for tinnitus, vertigo, and brain fog, there are not ostensible or clinically measurable manifestations of the disease. Not displaying something such as facial paralysis, often errantly called Bell's Palsy by physicians (Scorza and Finisterer, 2021), leads the clinician to order blood tests and perhaps an nMRI scan. Nothing will be found when the physician orders these tests. Using standard clinical measures without a semi-shotgun approach to assaying the patients exoproteosome, in search of autoantibodies, the clinician will find no problems with their patients and tell the patient, "It's all in your head" (Boodman, 2018). In terms of doing research on vaccine induced injuries, according to an article in Science, "I've talked to a lot of clinicians and researchers at various universities, and they don't want to touch it" (Couzin-Frankel and Vogel, 2022).

According to the CDC, among 71,491 U.S. adults who were hospitalized with COVID-19, 27.8 percent were overweight and 50.2 were obese (Kompaniyets et al, 2021). The analysis included 148,494 patients who received a COVID-19 diagnosis at emergency departments or inpatient visits between April 1 and Dec. 31 across 238 hospitals. Thus, 78% of the people sampled in this study who were hospitalized because of Covid-19, 78% were overweight or obese. Those who were overweight or obese were more likely to require invasive mechanical ventilation. Obesity was also linked to increased risk for hospitalization and death, especially among those under age 65. As BMI rose, so did the risk, the CDC found. Obesity is a major risk factor for autoimmune disease (Versini et al, 2014), and obese patients with Covid-19 produce a majority of SARS-CoV-2-specific antibodies that are autoimmune and not neutralizing (Frasca et al, 2021). Among other problems, autoantibodies can destroy neutrophils (Shastri and Logue, 1993) and leave the patient with a diminished innate immune response to the virus.

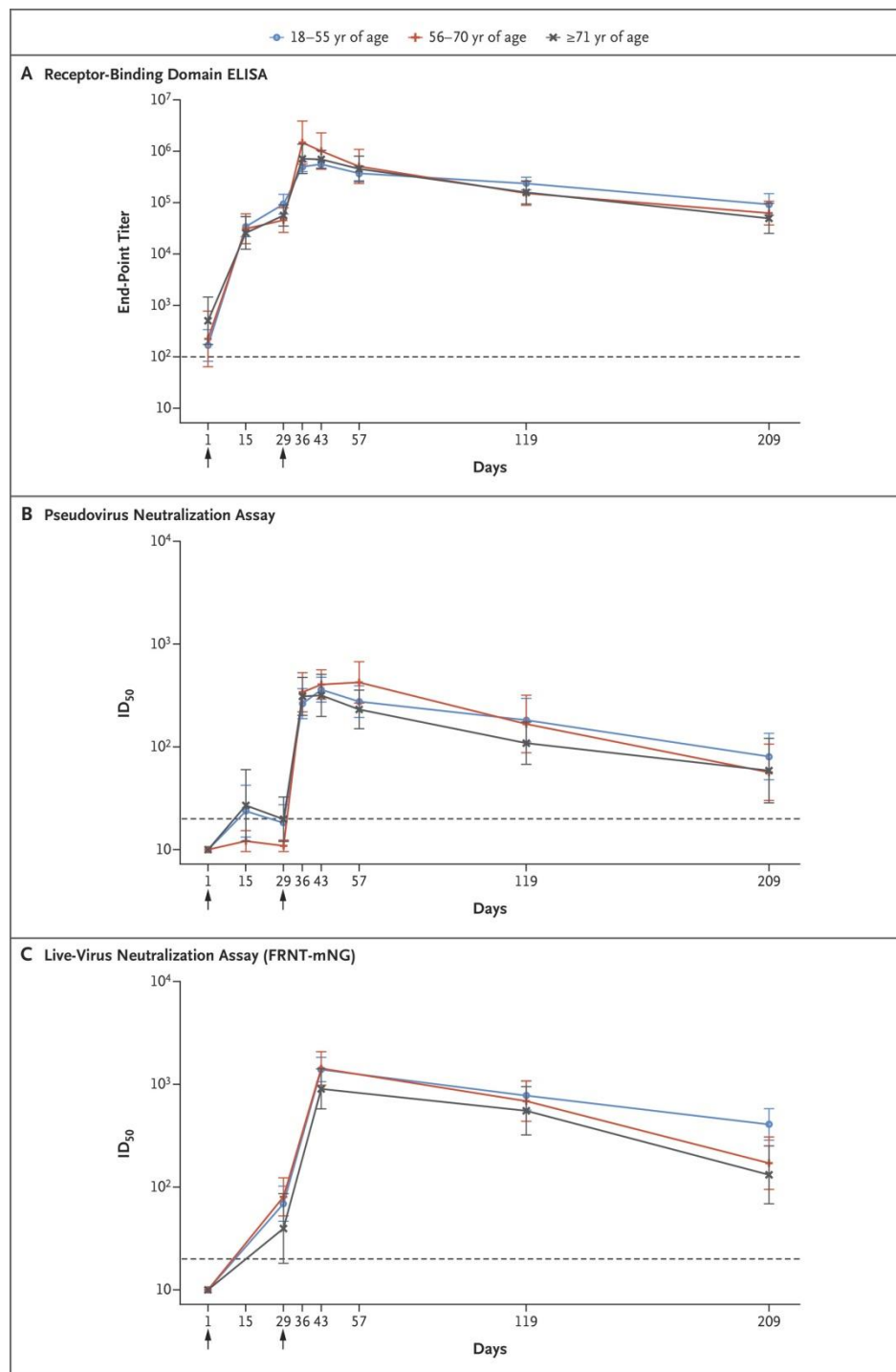


Figure 1. Time Course of SARS-CoV-2 Antibody Binding and Neutralization Responses after mRNA-1273 (Moderna) Vaccination. From Doria-Rose et al, 2021). The time course of autoantibody levels in the sera and other parts of the body may also be described by these graphs. For myself, the onset and exacerbation of my vaccine induced autoimmune disease from the mRNA vaccine has coincided with these curves where the onset of symptoms was a couple of day following the 2nd injection and steadily increased in severity over the next month. The symptoms have maintained for

10 months, but have been mitigated by a strict diet, including a low-fat, whole food plant based diet with no added salt, no gluten, and no emulsifiers. Reintroducing those ingredient exacerbates the symptoms.

Case Report

Treatment of life threatening autoimmune disease following Covid-19 vaccination has used a polypharmacy approach, directed at clearance and reduced production of autoantibodies. For example, IgA producing Beta cells in the gut play a key role in reducing inflammation (Rojas et al, 2019). In people treated for multiple sclerosis (MS), rituximab lowers immune B cell counts and reduces autoantibodies and autoimmune disease. The treatment relieved inflammation and reduced the risk of relapse. However, the drug, atacicept, that destroys even more B cells than does rituximab had the opposite effect to rituximab, triggering relapses. The evidence suggests that rituximab was efficacious because it destroyed the particular B cells that make the inflammation-causing immunoglobulin G antibodies. However, atacicept destroyed a different population of B cells found predominantly in the lining of the gut. These B cells, activated by bacteria, produce interleukin 10 (IL-10) that reduce inflammation. These gut-microbe-activated B cells may have been reducing MS symptoms through IL-10 activation, and therefore, the gut B cells destroyed by atacicept needed to be preserved by using rituximab. And we know that gut-associated IgA+ B cells and plasma cells traffic to the CNS and are part of the disease sequelae in MS (Probstel et al, 2020). However, rituximab doesn't stop the actions of intracellular autoantibodies that affect many targets and cause many symptoms of autoimmune disease (Burbello et al, 2021), including targeting the cytoplasmic domain of the $\beta 4$ integrin subunit in epithelial and mucosal cells (Bhol et al, 2000). Importantly, rituximab targets the CD20 antigen on B-cells, but not all B-cells express CD20 (Kuijpers et al, 2010), and therefore autoantibodies can still be produced by these non-CD20 B-cells. Perhaps these non-CD20 B-cells release autoantibodies that target intracellular targets. CD-20 appears to be regulated in B-cells (Pavlasova and Mraz, 2020), and therefore during conditions of an autoimmune response, may be down regulated in those cells releasing intracellular autoantibodies. These monoclonal antibody therapeutics have significant negative side

effects, including that some patients will never regain their B-cell compartment (Stensland et al, 2021).

As explained by Murphy and Longo (2021) a specific type of autoantibody is called an anti-idiotypic or antigen-binding domains, of some of the resulting anti-idiotypic (or “Ab2”) antibodies that are specific for Ab1 can structurally resemble that of the original antigens themselves. Thus, the Ab2 antigen-binding region can potentially represent an exact mirror image of the initial targeted antigen in the Ab1 response, and Ab2 antibodies have even been examined for potential use as a surrogate for the antigen in vaccine studies. However, as a result of this mimicry, Ab2 antibodies also have the potential to bind the same receptor that the original antigen was targeting. Ab2 antibodies binding to the original receptor on normal cells therefore have the potential to mediate profound effects on the cell that could result in pathological changes, particularly in the long term — long after the original antigen itself has disappeared. Ab2 antibodies can mediate the neurological effects of SARS-CoV-2 infection or vaccines, because ACE2 is expressed in neural cells, including neurons (Doobay et al, 2007), and the specific neuropathological effects of SARS-CoV-2 infection, and the similarity of these effects to Ab2-mediated neurological effects observed in other viral models.

Autoantibodies in Neurological Disease

Numerous autoantibodies types can target different cells and their components within the nervous system, leading to wide array of symptoms (Pruss, 2015). For example, myelin oligodendrocyte glycoprotein (MOG) autoantibodies target the myelin sheath of axons and induce FcR-mediated antibody-dependent cellular cytotoxicity (Brilot, 2009). Further, autoantibodies may target the myelin of the 8th cranial nerve, leading to vestibular dysfunction (Girasoli et al, 2018). In my case, I had symptoms of tinnitus, vestibular dysfunction such as dizziness, vertigo, and loss of balance, along with nystagmus and therefore degraded visual acuity. No hearing loss at any frequency was observed. The constellation of symptoms suggested that the vestibulocochlear nerve was targeted, possibly the myelin (Di Pauli and Berger, 2018). Autoantibodies against human epithelial cells and endothelial cells have been found to develop after SARS-

CoV infection (Yang et al, 2005). In my case, long-term blistering of the gums and mouth cavity has occurred for many months, and the symptoms spike at times and are correlated with spikes of the symptoms associated with autoantibodies targeting the 8th cranial nerve. Elevated frequencies of IgD–CD27– double negative B cells (DN B cells) with pro-inflammatory characteristics could be triggered because prolonged inflammatory cytokines, IL-6, IL-15, IL-8 (Frausen et al, 2019). This autoantibody type has frequently been found proximally at the site of lesions, including in the brain and CSF of MS patients (Baranzini et al, 1999; Qin et al, 2003). While the targets of antibodies released from these cells has not been described, immune reactivity against key myelin protein types by autoantibodies has been described in autoimmune patients (Mazón-Cabrera et al, 2019).

Dietary Remediation

The humoral immune response in the gastrointestinal tract is mediated by IgA+ memory B cells and IgA-producing plasma cells (activated B cells) in the gut-associated lymphoid tissue (GALT). Commensal and symbiotic bacteria act as critical B cell stimuli, playing an important role for the maturation of the GALT and further induce IgA production by B cells. The interrelation of dietary components, microbiome and B cell function are critical to the production of (auto)antibodies (Petta et al, 2018). Therefore, diet is critical to controlling autoantibody production, in turn, controlling the symptoms of autoimmune disease.

Reduce or Eliminate

Humans evolved eating mostly plants (Barras, 2016; Dunn, 2012), providing many benefits to the immune system (Jensen et al, 2021), including for better Covid-19 outcomes (Kim et al, 2021; Maguire, 2020). Animal products on the other hand, such as dairy, can lead to autoimmunity where lactose (Chiu et al, 2016), the bovine milk protein casein, and other milk proteins, have been found to target human glycoproteins and destroy myelin (Vojdani, 2014; Chunder et al, 2022). Plant foods are the sole source of dietary fiber, vitamin C, flavonoids, chlorophyll, and good sources of vitamin B1, folic

acid, potassium, and magnesium. They are also good sources of omega-3 fatty acids and their precursor molecule, alpha-linolenic acid (ALA), low in saturated fat, and do not contain cholesterol. As an example of the diet of early humans, sodium was solely derived from plants. (MacGregor and de Wardener, 1998), meaning that they ingested minute quantities of sodium. A high salt diet in modern man because of a diet of mostly processed foods and not whole plants causes disturbances in the ecological balance of the gastrointestinal microflora primarily through depletion of lactic acid-producing bacteria in a dose-dependent manner (Hamad et al, 2022). Since moderate increases in salt intake has proven to affect human immune cells, including T cells in vivo (Wilck et al, 2017; Willebrand and Kleinewietfeld, 2018), more specific analysis is needed to establish the role of NaCl in human autoimmune disease (Arroyo Homero et al, 2020), especially at low NaCl concentrations. Further, plant-derived nutrients have been found to be associated with an anti-inflammatory state by acting as ligands of the aryl hydrocarbon receptor (Jorg et al, 2016). AhR acts as a transcription factor in a variety of immune cells, including Th17 and Tregs, and has been associated with susceptibility as well as prevention of autoimmune diseases depending on its ligands. For example, indole-3-carbinol, deriving from crucifers such as broccoli, has been shown to suppress the production of proinflammatory cytokines, whereas the tryptophan-derived AhR ligand FICZ (6-Formylindolo(3,2-b)carbazole) derived from animal products at very high levels specifically increases the Th17 population and, therefore, worsens experimental autoimmune encephalomyelitis (EAE) severity. And chlorophyll ingestion from plants has many benefits beyond its well described anti-cancer properties (Dashwood, 2021), including anti-inflammatory (Subramoniam et al, 2012), and anti-viral properties (Liu et al, 2020). Chlorophyll also plays a role in regenerating CoQ10 (Qu et al, 2013). CoQ10 is an endogenous compound that acts as an antioxidant by scavenging free radicals, protecting our cells from DNA and protein damage (Pala et al, 2018).

Along with reducing or eliminating salt, eliminate wheat and milk because the peptide sequences of foods such as milk and wheat are similar to those of human molecules, such as myelin oligodendrocyte glycoprotein (Vojdani, 2015). Sprouted wheat has 47% less gluten (Boukid et al, 2017), and sourdough bread has less gluten (Thiele et al

(2004) than standard wheat, but they still contain gluten and can trigger autoimmunity. For myself, I don't eat dairy, but every time I eat wheat, my symptoms start to exacerbate several hours after the wheat consumption. Wheat, and other gluten containing grains, once ingested, gluten is partially cleaved into gliadin peptides that pass through the intestinal mucosa epithelial barrier due to increased permeability, caused by the inflammatory innate immune response. In the lamina propria (the intermediate connective tissue layer of the intestinal mucosa) occurs an important step in CD pathogenesis, gliadin deamidation by the tissue transglutaminase enzyme, which launches the activation of the adaptive immune system. This adaptive immune response against gliadin involves antigen-presenting cells such as macrophages, dendritic cells, and B cells. The innate immune response to gliadin occurs in the intestinal mucosa epithelial layer and increases the release of cytokines, namely interleukin-15 (IL-15), produced by enterocytes, macrophages, and dendritic cells. This results in intraepithelial leukocyte differentiation into CD8+ cytotoxic T cells that express the marker for natural killer NK-G2D cells causing epithelial cell apoptosis. The accumulation of all these inflammatory mediators leads to intestinal mucosal injury that manifests through flattening of the villi and elongation of the crypts, the histological alterations characteristic of CD. If one chooses to eat cheese, make sure that the cheese doesn't contain bacteriophage, either purposely added to control bacteria growth (Tabla et al, 2022) or through contamination (Atamer et al, 2013), because ingestion of bacteriophage may cause autoantibody production (Riley, 2004)

Reduce fat consumption because lipid consumption increases autoantibody production and autoimmune disease (Levy et al, 1982; Winer et al, 2011; Pham et al, 2017), and leads to systemic, chronic inflammation (Duan et al, 2018).

Eliminate meat consumption (Jin et al, 2021; Samraj et al, 2014; Bashir et al, 2020). Selenium (McLachlan et al, 2017), and iodine (Burek and Yaylor, 2009) have been found to increase autoantibody production.

Add or Increase

A few examples of why adopting a whole food plant based diet includes, a diet rich in short-chain fatty acids (SCFAs) could positively impact gut microbiota and inflammatory processes (Jorg et al, 2016). The microbiome converts non-digestible carbohydrates from plants (dietary fibers) to SCFAs, including acetate, butyrate, and propionate, which reduce the risk of inflammatory diseases, type 2 diabetes, obesity, heart disease, and other conditions. Tea contains polyphenols, and is easily and inexpensively consumed by most people (Winiarska-Mieczan et al, 2021). The immunomodulatory properties of polyphenols may, in turn, be useful in alleviating the symptoms of autoimmune disorders. Polyphenols are capable of activating intracellular pathways (e.g., the arachidonic acid-dependent pathway, the nuclear transcription factor (NF- κ B), mitogen-activated protein kinases (MAPK), phosphatidylinositol 3-kinase/B protein kinase signaling pathway (PI3K/Akt) as well as stimulating epigenetic modulations that regulate the organism's immune response. Ginger reduces autoantibody production (Ramadan et al, 2020), and a cocoa-rich diet decreases autoantibody production, conferring beneficial immune function (Camps- Bossacoma et al, 2017).

Discussion

Quoting from Scorza and Finisterer (2021), "Real world data rather indicate that the spectrum of side effects to any of the commercially available SARS-CoV-2 vaccinations is broader than anticipated, underreported, and played down. Side effects need to be thoroughly elaborated to draw more real pictures than those frequently sold. Real world is more unsafe than its propagated image." The role of vaccines in inducing autoimmune disease needs to be studied (Chen et al, 2001; Toussirost and Bereau, 2015; Principi and Esposito, 2020; Chen et al, 2022).

If we look closely at the genomes of the coronaviruses that have emerged from bats and other species, we see that these viruses can readily recombine amongst each other, in addition to the point mutations we have seen in Omicron and in other known SARS-CoV-2 variants. Recombination, we know from influenza, can lead very quickly to

much more virulent variants by picking up components that our immune systems have not previously seen. SARS-CoV-2 is mutating and recombining rapidly to form new variants, with some of the variants becoming able to evade the vaccinated immune system (Pulliam et al, 2021). A “booster” vaccine against the new Omicron variant is now being designed. Instead of a vaccine inducing a supranormal antibody response measured in the blood, broader immunity induced in the respiratory tracts as well as the blood has been suggested as a new vaccine strategy (Maguire, 2022). This would mean a vaccine that induces a broader immune response than that simply aimed at the spike proteins, and delivering the vaccine IM as well as intranasally. This would elicit an immune response throughout the body, including the respiratory tracts where the virus first infects and replicates, and not simply a huge spike of antibodies in the blood. In this manner, autoantibody production may be limited relative to a supranormal antibody response elicited from a large antigen introduction to only the muscle.

The incidence of autoimmune diseases ADs, approximately 3–5% worldwide, is increasing in westernized societies, as confirmed by epidemiological studies; these suggest that multiple sclerosis (MS), type 1 diabetes (T1D), inflammatory bowel diseases (mainly Crohn’s disease), systemic lupus erythematosus (SLE), primary biliary cirrhosis, myasthenia gravis, autoimmune thyroiditis, hepatitis, and rheumatic diseases are steadily increasing. The geo-epidemiological distribution of ADs, their correlation with socioeconomic status, and their rapid increase in developed countries, together with observations in migrant populations, suggest that environmental factors, rather than genetic ones, are chiefly driving these evolutionary processes (Mazzuca et al, 2021). Part of the mechanisms underlying the increase in ADs throughout Westernized societies over the last three decades may be explained by the increased intestinal permeability induced by industrial food additives (Fasano et al, 2005). Vaccine induced autoimmune disease needs to be studied at multiple levels, including epidemiologically to understand its incidence and those who are susceptible, along with mechanistic studies to understand processes of the diseases, and prevention and treatment of vaccine induced autoimmune disease.

Conclusions

Autoimmune disease following mRNA vaccination for Covid-19 is not well understood and is underreported. For severe, life-threatening cases, a polypharmacy approach is required to remediate the severe reaction. For less severe cases where autoantibodies are implicated, regression to the mean as the sera antibody titers fall coupled with dietary measures will greatly diminish the symptoms, including tinnitus and vestibular dysfunction.

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