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Vaccine Induced Autoimmunity May Cause Autism and Neurological Disorders

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Abstract

Much evidence has accumulated that vaccines not only cause inflammation but also induce autoimmunity. While the efficacy for many vaccines has been demonstrated in some form, such as prevention of infection or reduction in severity of symptoms, the safety profile, especially for the long-term, has not been well studied. Further, the long-term efficacy of some vaccines is worse than normal immunity. All drugs, including vaccines, need thorough studies of risk versus benefits. For many drugs, the risks, and especially the long-term risks, have not been thoroughly studied and therefore the risk versus benefit of the vaccine is not understood. Long-term scientific studies from non-sponsors performed by non-conflicted scientific groups in academia are needed to evaluate the long-term safety of vaccines, and then the use of these data to develop safer and more efficacious vaccines can be accomplished.

Keywords: Vaccine, Inflammation, Immunity, Autoimmunity, Autism, Neurological Disorders

Introduction

Man did not evolve to encounter all the zoonotic viruses that have infected us in the modern world. While plant foods have been important in the evolution of our human ancestors (Ahituv et al, 2025), humans and animals have coexisted for millennia, and anthropogenic factors, such as raising and consuming cattle (McDaniel et al, 2014; Leibler et al, 2023; Garg et al, 2024), have greatly increased interactions between the two populations, thereby increasing the risk of disease spill-over. Of the nonillions of viruses in the world, there are only 9,110 named species listed by the International Committee on Taxonomy of Viruses (Vance, 2021). Many of the unknown viruses are likely infecting us or are capable of infecting us. The many serotypes of each virus strain, which confer many aspects of viral infection (such as Dengue; Bos et al, 2024), further increase the number and complexity of viruses that man encounters. Increasingly the strategy of modern society has been to increase the number of vaccine types and to repeatedly "boost" the different types of vaccines over a person's lifetime. Such strategies have consequences. Antibody-dependent enhancement of viruses due to vaccination is one of many negative side-effects that vaccines cause (Wan et al, 2020). For example, maternal derived–antibodies can lead to ADE in children (Katzelnick et al, 2017), increasing the probability of viral infections in the rapidly developing and vulnerable child. Vaccine induced ADE (Antibody-dependent enhancement) is well established in viral infections (Ikewaki et al, 2023) but has an underappreciated role during bacterial, fungal and parasitic infections (Wells et al, 2025). Cross-reactive antibodies associated

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with different viruses and their vaccines at sub-neutralizing concentration may lead to ADE for a virus other than that associated with the original virus or infection (Thomas et al, 2024). Measuring neutralizing antibody (nAb) titers against viruses has not always correlated with protection in natural infection studies and vaccine trials (Dias et al, 2024), and cross reactivity of nAb can mean that one vaccination may lead to ADE for other virus types even where nAbs have been measured. Further, given that multi-ohmic studies of human brains have found a peak susceptibility to autism during the second trimester (Wang et al, 2025) and that the maternal passage of vaccine related molecules to the fetus following vaccination of the mother in her second trimester regularly occur, studies of the fetus are needed. Given the likelihood of viruses originating from cells (Nasir et al, 2020) and therefore the continued origins of novel viruses from modern cells, how many new vaccines with their associated adverse events will man have to administer for protection against the subset of nonillions of viruses that threaten us? Further, when considering "original antigenic sin," where we consider "first flu is forever," the imprinting of so many vaccines may render the immune system incapable of neutralizing new viruses or new strains of viruses (Monto et al, 2017). Enhancement of infection and subsequent cross reactivity is an important phenomenon in autoimmune conditions, possibly of the brain, including demyelinating conditions such as myelin oligodendrocyte glycoprotein (MOG) autoimmunity (Schanda et al, 2024). Other strategies in combination with vaccines are warranted (Maguire, 2020; Maguire, 2021).

Limitations of Vaccines

As Steir et al (2025) have written, "Questions about vaccine safety and efficacy are not just valid – they're essential." But Steir et al (2025) go on to debunk some of the publications, often in non-peer reviewed publications, that question vaccine safety and efficacy and sometimes call for vaccines to be pulled from the market. My intent here is to question vaccine safety and efficacy so that rational dialogue can be had and further research implemented to improve vaccine safety and efficacy. As an example, I have previously published a paper in a vaccine journal offering a means to improve Covid-19 vaccine safety and efficacy (Maguire, 2022) and also offered a new means to implement a vaccine for upper respiratory infections, such as Covid-19, without presentation of antigen (Maguire, 2021). Wanting to improve vaccines is not anti-vax, rather it is the nature of science to constantly improve our knowledge and then up to society, including our money-driven (Stevens and Glatstein, 1996), woeful medical system (Lyu et al, 2017; Trasta, 2018; Berwick, 2023) that is full of flawed drugs (Langreth et al, 2024), to implement what scientists have learned. Nordstrom et al (2022) published a study in the Lancet tracking the effectiveness of COVID-19 vaccines over time. They found that vaccine waning began after 2 months and once eight months had elapsed since the second injection of the two-injection series, immune function was lower than that among unvaccinated individuals (see Fig.2), and the authors therefore recommended a third dose. A recent study in Austria from the University of Graz (Riedmann et al, 2025) has found the population maintains a high level of post-pandemic protection against COVID-19 deaths (however, other health parameters were not measured), irrespective of vaccine boosters. Repetition of other vaccines has also been found to reduce immunity (Ohmit et al, 2014). Vaccination may intensify the disease process for certain infections, including dengue and respiratory syncytial virus, or may increase vaccine resistance (Majumder and Razzaque, 2022). Dengvaxia vaccination has been found to enhance the dengue infection and intensified the disease process, given vaccination failed to induce cellular immunity against dengue virus. As Muturi-Kioi et al (2016) have reported, "Post-vaccination neutropenia is not uncommon, generally transient and clinically benign, but many vaccine trials do not have a sampling schedule that allows its detection."

While vaccines can be very efficacious, long-term studies are warranted. Further, boosters can temporarily restore higher levels of antibodies (and autoantibodies), frequent boosters may further erode innate immune function, such as neutropenia (Muturi-Kioi et al, 2016), leading to an increased risk of various infections and cancers. Stratton et al (1994) published the first report on a causal relationship between several vaccines, diphtheria, tetanus toxoids, oral polio vaccines, and autoimmune disorders, such as Guillain–Barre syndrome (GBS), type 1 diabetes, and multiple sclerosis, and more recently, thyroid eye disease (Muller et al, 2024). GBS is a rare, acquired demyelinating polyneuropathy that causes muscle weakness, often beginning in the lower extremities and ascending over time with loss of reflexes. It sometimes leads to paralysis. In January 2025, the FDA issued a safety communication that warns about possible GBS with respiratory syncytial virus (RSV) vaccines manufactured by Pfizer (Abrysvo) and GSK (Arexvy). These vaccines are approved for pregnant mothers and young children and their consequences in the fetus are unknown.

In September 1993, the Institute of Medicine released a report entitled Adverse Events Associated With Childhood Vaccines: Evidence Bearing on Causality. The committee found that the evidence favored acceptance of a causal relation between diphtheria and tetanus toxoids and Guillain-Barré syndrome and brachial neuritis, between measles vaccine and anaphylaxis, between oral polio vaccine and Guillain-Barré syndrome, and between unconjugated Hib vaccine and susceptibility to Hib disease. The committee found that the evidence established causality between diphtheria and tetanus toxoids and anaphylaxis, between measles vaccine and death from measles vaccine-strain viral infection, between

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measles-mumps-rubella vaccine and thrombocytopenia and anaphylaxis, between oral polio vaccine and poliomyelitis and death from polio vaccine-strain viral infection, and between hepatitis B vaccine and anaphylaxis.

Recent studies have found that mRNA vaccinations induce a high proportion of spike-specific IgG4 antibody responses, which is associated with autoimmune disease and specifically autism. As Vadala et al (2017) have written, "vaccination might display autoimmune side effects and potentially even trigger a full-blown autoimmune disease." For many drugs, the risks, and especially the long-term risks, have not been thoroughly studied (Resnick, 2007). This is especially true of vaccines (Block, 2023). Given the evidence presented in this review, I recommend thorough, long-term scientific studies from non-sponsors performed by non-conflicted scientific groups in academia to evaluate the long-term safety of vaccines and using these data to develop safer and more efficacious vaccines (Maguire, 2022). This can only happen through government directives and monetary support.

Most of the studies on vaccine-related autoimmune adverse events are woefully inadequate and reduce to a 10–20-day follow-up analysis, using the recruitment of putatively linked autoimmune events (Vadala et al, 2017). In many developed countries, by the time children are 4 to 6 years old, they will have received a total of 126 antigenic compounds along with high amounts of aluminum (Al) and thimerosal adjuvants through routine vaccinations. The evidence regarding adjuvants such as thimerosal and aluminum was indirect and incomplete regarding neurodevelopmental disorders for years (Stratton et al, 2001), until recent studies detailed some of the associated injuries (Exley and Clarkson (2020). Many of the excipients, including adjuvants, are listed online by Johns Hopkins University (https://www.vaccinesafety.edu/components-excipients/). As McKee et al (2007) have published, "Despite its [aluminum] long use, we still do not know exactly how alum mediates its adjuvant effects." Even preterm babies, who's brain and nervous system is less developed and more susceptible to environmental perturbations than babies born at full-term (Bouyssi-Kobar et al, 2016), are routinely given vaccines at birth or shortly after birth (Sadeck and Kfouri, 2023). Movsas et al (2013) in JAMA Pediatrics report that, "an infant at the 2-month checkup receives multiple aluminum-containing vaccines that in combination may have as high as 1225 µg of intramuscular aluminum; this is a much higher intramuscular aluminum dose than the safely recommended intravenous aluminum dose."

Not only are the clinical trials too short to measure autoimmune events, the oversight by the FDA of clinical trials is "grossly inadequate" (DeMassi, 2022), and the production of vaccines is not properly monitored for safety (Hernandez and White, 2021; Chooi et al, 2022), and likely worse under presidential administrations, such as Trump, that value deregulation (Piller, 2019). For example, even when drugs approved in the USA are studied in the short term, most cancer drugs granted accelerated approval did not demonstrate benefit in overall survival or quality of life. Fewer than half (20/46, 43%) of the cancer drugs demonstrated a clinical benefit in confirmatory trials (Liu et al, 2024). As Halvorson (2007) has published about the USA healthcare system, "We don't really have a health care delivery system in this country. We have an expensive plethora of uncoordinated, unlinked, economically segregated, operationally limited micro systems, each interacting in ways that too often create suboptimal performance both for the overall health care infrastructure and for individual patients." Vaccine programs in the USA reflect this, as Halvorson writes, "perversely incented" healthcare system.

Neural Effects

Numerous reports of neural injuries (Schofield and Hendrickson, 2018), such as demyelination, are documented in peer reviewed publications. The most common postvaccination demyelination is acute disseminated encephalomyelitis, but cases of optic neuritis, transverse myelitis, and multiple sclerosis relapses have been reported (Kumar et al, 2020; Simone et al, 2021; Jarius et al, 2022). Recently, an increasing number of postvaccination neuromyelitis optica spectrum disorder (NMOSD) cases have been reported. Kumar et al (2020) report an unusual case of myelin oligodendrocyte glycoprotein antibody–related NMOSD after multiple vaccines in a first-trimester pregnant woman. Health status of the fetus was not reported – did autoantibodies affect fetal development of the brain? Aratani et al (2016), using a murine model, found that HPV-vaccinated donners are susceptible to the HPV vaccine and may develop HPV vaccination associated neuro-immunopathetic syndrome (HANS) under certain environmental factors. A class action lawsuit against Gardasil manufacturer Merck has been filed. Myelin oligodendrocyte glycoprotein antibody-associated disease has also been reported for Covid-19 vaccination (Morena and Gyang, 2022), and a lawsuit has been filed against Pfizer, one of the manufacturers of mRNA Covid-19 vaccines. Vaccines may trigger a preexisting latent autoimmune disease. For example, this was reported in a case of neuromyelitis optica spectrum disorder (NMOSD) involving a patient that was found to be seropositive for the AQP4-IgG over 10 years before the onset of NMOSD (Nishiyama et al, 2009). Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disorder (MOGAD) is an autoimmune demyelinating disorder that is often associated with acute disseminated encephalomyelitis and usually occurs postinfection or postvaccination. MOGAD after mRNA severe acute respiratory syndrome coronavirus

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2 (SARS-CoV-2) vaccination has been reported in healthy 68-year-old woman (Matsumoto et al, 2022). Similar reports of MOGAD for the non-mRNA Covid-19 vaccines have been reported (Francis et al, 2022).

Considering infection and/or vaccination (Meltzer and Van de Water, 2016), some cytokines induced by either can inhibit neurogenesis and promote neuron death, while other cytokines can promote the growth and proliferation of neurons and oligodendrocytes. Induction of complement proteins and immune responsive microglia from infection or vaccination can participate in synaptic scaling and pruning, while brain-reactive autoantibodies can change the development and function of neurons. When some of the many components of the immune system are dysregulated, these immune responding networks can lead to abnormal changes in neurodevelopment and behavior. For example, vaccination can induce autoimmunity (Orbach et al, 2010), which increases the prevalence of shingles (Yun et al, 2016), which is associated with autism (Gentile et al, 2014). We know that many infections of the mother can infect or otherwise harm the fetus (Auriti et al, 2021). The first suggestion of a link between the immune system and Autism Spectral Disorder (ASD) was by Stubbs (1976) because of undetectable rubella antibody titers after a rubella vaccine challenge in autistic children. Several animal studies confirmed that an immune challenge during pregnancy resulted in behavioral abnormalities. Maternal immune challenge was found to activate multiple inflammatory pathways, including a macrophage inflammatory state that increased M1 polarization (Onore et al, 2014), leading to up-regulation of interferon-gamma (IFN-γ) and interleukin (IL) 17a secreted by CD4+ T cells (Luan et al, 2015), and cause a permanent, systemic deficit of T regulatory cells (Tregs) (Hsiao et al, 2012). Cheng et al (2017) found that exosomes from M1-polarized macrophages potentiated a cancer vaccine by creating a pro-inflammatory microenvironment in the lymph node. Thus exosomes from polarized M1 macrophages due to vaccination can have widespread pro-inflammatory effects, and can pass inflammatory signaling from mother to fetus (Sheller-Miller et al, 2019).

Imperfect vaccines and the evolution of pathogen virulence

Considering evolution, instead of just asking what genetic mutations might give one biological entity an advantage over others, evolutionary biology should also focus on the developmental mechanisms and structures that underlie fitness differences (Layland et al, 2015). Most vaccines do not provide full protection from disease, and are therefore instructive to as what will evolve. These partially effective (imperfect) vaccines may be used to protect both individuals and whole populations. Gandon et al (2001) studied the potential impact of different types of imperfect vaccines on the evolution of pathogen virulence and the consequences for public health. They found that vaccines designed to reduce pathogen growth rate and/or toxicity diminish selection against virulent pathogens. The viral evolution leads to higher levels of intrinsic virulence and hence to more severe disease in unvaccinated individuals. This viral evolution can erode any population-wide benefits such that overall mortality rates are unaffected, or even increase with such vaccines, with the level of vaccination coverage. Read et al (2015) further tested this hypothesis in chickens. Chickens were infected with Marek's disease virus of different strains known to span the spectrum from low to high virulence. In those birds that weren't vaccinated, the infection with highly virulent strains killed them so fast that they shed very little virus. The effect was orders of magnitude less than when they were infected with less virulent strains. However, in vaccinated chickens, the opposite was true – those chickens infected with the most virulent virus strains shed more virus than birds infected with the least virulent strain. Thus, "vaccination enabled the onward transmission of viruses otherwise too lethal to transmit, putting unvaccinated individuals at great risk of severe disease and death" (Read et al 2015).

As Read has said (Kupferschmidt, 2015), "We are entering the era of leaky vaccines in humans." Candidate vaccines against Ebola or malaria—one of which recently received an important stamp of approval in Europe—should definitely be used if they are safe and effective, he says, but they could lead to more virulent pathogens. "We need to have a responsible discussion about this. "Boots (2015) writes, "The Read paper has shown, however, that this piece of evolutionary theory is pertinent to real-world infectious disease control. More generally, this study highlights the potential usefulness of evolutionary theory for disease control and suggests that it may therefore have an important role to play in the design of medical interventions." Thus, increased virulence due to vaccination may lead to longer and more severe infections, increasing the odds of developing autoimmunity (Uversky et al, 2023).

Immune Modulation Affects the Germline

Exosomes are known to travel to the germline where environmental experience could effect a transmissible epigenetic change in the germline (Eaton et al, 2015; Noble, 2020; Chen et al, 2023; Wong et al, 2024). Similarly, exosomes from T-cells are known to induce inflammation (Shao et al, 2021). Thus, any immune response, whether innate and/or adaptive, induced by vaccination may lead to germline changes and possible inherited disorders. Beyond epigenetics, instability of the genome itself in germ cells can be induced by inflammation (Ma et al, 2024). For example, germline mutations in PTEN, the gene that encodes

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phosphatase and tensin homolog, have been detected in about 20% of children with autism spectrum disorder (ASD) and macrocephaly and are associated with marked abnormalities in the white matter of the brain (Busch et al, 2019). As Jung et al (2024) have stated following an epidemiological study of autoimmune-connective tissue disorders (AI-CTDs), "given the indolent course of AI-CTDs, these results suggest that long-term surveillance for the development of AI-CTDs after mRNA vaccination may be warranted." Connective tissue inflammation is often manifest in the nervous system (Poshattiwar, 2023).

Maternal Immune Modulation Affects the Fetus – Placental Translocation

Various nano-sized exogenous materials such as aluminum (a vaccine adjuvant), gold, silica, plastic, and titanium dioxide have been shown to cross the placental barrier and exert toxicity in animal studies (Anane et al, 1997; Medley et al, 2023), along with various endogenous materials, such as maternal immunoglobulins and inflammatory cytokines. The placenta itself can become inflamed (Thornburg and Marshall, 2016), leading to decreased transport of necessary nutrients to the fetus (Zhang et al, 2016). The placental transfer of maternal immunoglobulin to the developing fetus is a specific adaptive mechanism that confers short-term immunity in the neonate by providing the immunologically naïve fetus with a subset of the maternal humoral immune system, including IgG, specifically IgG4 subtypes (Palmeira et al, 2012). Numerous autoimmune disorders have been identified, and nearly 80% of these disorders are encountered in women of childbearing age (Borba et al, 2019). Thus, some conditions such as maternal autoimmune disorders or maternal immune responses against paternally inherited fetal antigens, the transfer of antibodies can have a deleterious effect upon the fetus with potential long-term consequences (Chiobanu et al, 2020).

The presence of maternal anti-fetal brain IgG autoantibodies has been reported to play a role in ASD (Fox-Edmiston et al. (2015). Monkeys exposed prenatally to human immunoglobulin G (IgG) derived from mothers of ASD children were found to exhibit stereotypies, hyperactivity (Martin et al, 2008), or impaired social behavior (Bauman et al, 2013). As an example of how powerful a vaccine can be to the fetus, the majority of infants born to COVID-vaccinated mothers had persistent, elevated anti-S antibodies at 6 months compared with infants born to mothers with SARS-CoV-2 infection (Schook et al, 2022). Antibodies are well known to cross the placental barrier, and fetal effects of maternal mAb biologic treatment have been reported (Genevieve et al, 2018). For example, infants exposed to maternal rituximab, an anti-CD20 mAb, have been reported to have low B cell numbers, non-detectable circulating IgG levels, and potential infection complications. Neonatal lupus erythematosus has been found to be associated with breast milk autoantibodies, IgG and IgA anti-nuclear and anti-Ro antibodies (Gray et al, 2007). Discontinuation of breastfeeding eliminated the inflammatory, autoimmune condition in the neonate's skin. Family history of autoimmunity has been reported as a risk factor for ASD in multiple studies (Gładysz et al, 2018). A meta-analysis on this topic identified hypothyroidism, type 1 diabetes, rheumatoid arthritis, and psoriasis as a major family history burden. Maternal autoimmune diseases beginning during pregnancy can strongly impact risk of ASD in offspring as well. Chronic chorioinflammation has also been associated with CNS white matter injury in newborns, but this increased risk was seen in newborns with chronic chorioinflammation and umbilical cord inflammation while neither condition alone was associated with white matter injury (Korzeniewski et al, 2014). This is another study suggesting that the interaction of mechanisms rather than one etiology may be responsible for initial neurocognitive pathologies.

The Vaccine Industry

In the past 20 years, the vaccine business, a former small growth sector in the pharmaceutical business, has shown remarkable growth powered by new vaccines for more diseases (Douglas and Samant, 2018). As part of the inducement to grow the vaccine industry, vaccine makers cannot be sued for any injuries that their vaccine may cause (Richey, 2011). Covid19 vaccines alone as of Feb 2023 have made $90 billion for pharma (da Hann, 2023) in this ever-growing industry. Revenue incentives increase the development of new vaccines, but not their needed safety studies (Classen and Classen, 1999; Federico, 2024). Vaccine injuries may be missed as a result (Mohseni et al, 2022), such as chronic intestinal inflammation (Alwali et al, 2024), a potential trigger of autoimmune disease (Halling et al, 2017). "More empathy—and investment—is needed to address vaccine-related injuries," says Harvard T.H. Chan School of Public Health's Kizzmekia Corbett-Helaire (https://hsph.harvard.edu/news/vaccine-injuries-deserve-more-attention-says-vaccinologist/). As Finsterer (2024) reports in a case study, "It is undisputed that anti-SARS-CoV-2 vaccines can have side effects. Long post-COVID vaccination syndrome (LPCVS) is one of them and is often neglected."

Childhood Vaccines and the Increasing Incidence of Autism

A child's first vaccines are usually given within 12–24 hours of birth. The first vaccine is typically for Hepatitis B (HepB). The childhood vaccine schedule in the USA includes 15 different immunizations (shots or drops). In the U.S., about 4 in 100 boys and 1 in 100 girls have autism. Boys are nearly 4 times more likely to be diagnosed with autism than girls. Prior to 2016, ASD prevalence was consistently higher

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among White children than other racial or ethnic groups (Grosvenor et al, 2024), and vaccination rates have been higher in White children compared to Blacks (Barker et al, 2006). According to recent data from the CDC, the prevalence of autism spectrum disorder (ASD) in the USA is on the rise, with the current estimate being that 1 in 36 children are diagnosed with autism, marking a significant increase from previous rates. This is a significant increase from the 2021 estimate of 1 in 44, which was a big jump from 1 in 110 in 2006, meaning the number of children diagnosed with autism is steadily climbing. Maenner et al (2023) report that the prevalence of autism among children in the US has risen over 4-fold in the past 2 decades, from 6.7 cases per 1000 (1 in 150) in 2004 to 27.6 per 1000 (1 in 36) in 2020. While ethnic minority and low-income children may be disproportionately harmed by exposures to neurotoxicants (Payne-Sturgess et al, 2023), they are less likely to be harmed by vaccines because of fewer vaccinations. Clearly, epidemiology illuminates the cause of autism as being a function of one's exposome, including possibly vaccines, or the parental exposome owing to transgenerational epigenetic inheritance.

What do I mean by that statement? Some will argue that vaccines can't cause autism because signs of autism begin in the fetus. But vaccines cause epigenetic changes (Bannister et al, 2020), and epigenetic changes can be inherited by the fetus (Choi et al, 2016). Further, epigenetic changes in the fetus can arise from what the mother is exposed to as she carries the fetus (Heard and Martinssen, 2014). And vaccines are routinely given to pregnant mothers (Swamy and Heine, 2015). So the limited evidence suggesting that autism begins in the womb before vaccines are administered to the fetus and therefore vaccines cannot be the cause of autism is a spurious argument. Epigenetic methylation changes due to vaccines (Bannister et al, 2020; Pang et al, 2022), including at cytosine-phosphate-guanine sites (CpGs) (Janjanam et al, 2016), that occur during early childhood may be associated with antigen-specific antibody responses to vaccines (Pischedda et al, 2021). Methylation of cytosine promotes C-to-T mutations (Beletskii and Bhagwat, 1996). A C-to-T mutation in the CNS is associated with autism, referring to a specific genetic change where a cytosine (C) nucleotide in the DNA is replaced by a thymine (T) nucleotide that can occur in genes associated with brain development and function. This may contribute to the development of autism by disrupting the protein produced by that gene; one prominent example is a mutation in the CHD8 gene, where such C-to-T changes can lead to significant disruptions in neural development and are considered a risk factor for autism (Bernier et al, 2014). A close coupling between the accumulation of sporadic somatic mutations and the widespread changes in methylation has been observed in humans (Koch et al, 2025).

General Manifestations of Vaccine Injuries

I'll first give an example of how vaccine candidates are injuring infants, and how these candidates continuously do so even though we've known for 50+ years that we don't understand how the vaccine candidates are injuring infants. Yet we continue administering such vaccines.

Poorly Designed mRNA Vaccines.

Modified ribonucleotides are commonly incorporated into therapeutic IVT (*in vitro* transcribed) mRNAs to decrease their innate immunogenicity, but their effects on mRNA translation fidelity are poorly understood. Scientists (Mulriney et al, 2024) have discovered that misreading of therapeutic mRNAs by the cell's decoding machinery can cause an unintended immune response in the body. They have identified the sequence within the mRNA that causes this to occur and found a way to prevent 'off-target' immune responses to enable the safer design of future mRNA therapeutics. Further, GC enrichment in the mRNA vaccines (Seneff et al, 2022; Kon et al, 2023) may lead to chronic RNA G-quadruplex accumulation (Wang et al, 2021), a stable RNA structure that modifies gene expression. G-quadruplex accumulation is a mechanism associated with aging brains and Alzheimer's (Kallweit et al, 2025).

Poorly Designed Polio Vaccines and the Myth of Salk and Sabin

The average number of paralytic polio cases each year in the USA was 16,316, and 1,879 people died from polio before the polio vaccine. In 1946, Joeseph Melnick, Ph.D. first isolated the polio virus (https://pmc.ncbi.nlm.nih.gov/articles/PMC435562/pdf/jcinvest00597-0123.pdf), and in1949, John Enders, Ph.D. and his team first cultured the polio virus (Enders et al, 1949). This set the stage for others to develop polio vaccines, and Enders then shared samples and techniques for tissue culture with Salk's team for vaccine development. As Salk said, "I was not trained as a scientist. I was trained in medicine." But a mythology developed, started and perpetuated by Salk himself that he was the inventor of the polio vaccine (Matysiak, 2005). Herald Rea Cox, Ph.D. avoided the technique developed by Dr. Enders because of the danger monkey virus represented. Dr. Enders used rhesus monkey kidneys and testicles to develop the cell culture technology. In October 1952, Cox reported that he had grown the Lansing strain of polio virus, the strain Dr. Ender's had cultured, in fertile hens' eggs, and in 1961, he announced an oral polio vaccine. Sabin, a physician, would then run clinical trials on the partially inactivated virus that Dr. Cox had cultured. But the Ender's vaccine, erroneously called the "Salk vaccine" didn't work well. Children were developing polio despite being vaccinated. While Ender's vaccine reduced the incidence of polio among middle-class Americans, its cost and its requirement of three injections

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and a booster meant that for years the disease continued to affect the poor and others lacking access to proper medical care. More importantly, Joshua Lederberg, Ph.D. has said that the formaldehyde used to inactivate the polio virus will disassociate from the inactivated complex, thus allowing the virus to restore its infectivity. This is what led to the "Cutter Incident" where the original polio vaccine caused 40,000 cases of polio, leaving 200 children with varying degrees of paralysis and killing 10 (Fitzpatrick, 2006).

Then Cox's vaccine, often referred to as the "Sabin's oral vaccine," which was cheap and easy to administer, was licensed for production in 1962, with the hope that polio could be fully controlled in the United States using Cox's oral vaccine. Along with an increase in autoimmune disease, Guillain-Barré syndrome (Kinnunen et al, 1989), the oral vaccine has caused many cases of polio-vaccine induced polio, until this day in some parts of the world, where vaccine-derived poliovirus type 2 has continued to circulate, paralyzing more than 3300 children (Roberts, 2024), and therefore the USA stopped using the oral vaccine in 2000. In other words, both the original inactivated polio vaccine (Ender's technology) and oral polio vaccine (Cox's technology) were flawed vaccines and paralyzed and killed many children. We were testing these vaccines in an unsuspecting population that believed the Salk and Sabin myths that were widely circulated by the media. To be clear, the oral polio vaccine (OPV), made of functional, weakened polioviruses, has been the most effective vaccine for eradicating polio because it induces strong immunity in the gut and spreads through the stool of immunized children, also protecting those who don't receive the vaccine drops. However the classic oral polio vaccine (OPV) no longer protected against one of the three types of poliovirus. Type 2 virus had been eradicated by 2016, and the only remaining type 2 polio cases were touched off by the live virus in the vaccine itself. Dropping the type 2 component from the polio vaccine was implemented to prevent these vaccine injuries from the type 2 virus.

But "the switch," as this global move has become known, became "an unqualified failure," according to a draft report commissioned by the Global Polio Eradication Initiative (GPEI) that is now open for public comments (Roberts, 2024). Unexpectedly, vaccine-derived poliovirus type 2 has continued to circulate after the switch, paralyzing more than 3300 children. Neurovirulent PV variants in OPV recipients is thought to be causal (Wimmer et al, 1993), and infect the brain in addition to motor neurons (Daley JK et al, 2005). And GPEI (Global Polio Eradication Initiative) has spent more than $1.8 billion trying to stop these outbreaks, mostly in Africa. Those numbers are likely to increase until the polio program finds a means to fix the problem. Some have called for stopping all use of the cheaper OPV. The price of OPV ranges from $0.12 to $0.18, substantially less than the $1.00 to $3.28 range for IPV (Alfaro-Murillo JA et al, 2020). In 2022, a petition (https://icandecide.org/wp-content/uploads/2023/05/Petition-IPOL-2022-08-23.pdf) was filed to the HHS to have the currently used IPOL polio vaccine (Poliovirus Vaccine Inactivated - Monkey Kidney Cell) removed from the market for infants and toddlers until a properly controlled and properly powered double-blind trial of sufficient duration is conducted to assess the safety of the product as required pursuant to applicable federal statutes and regulations for licensing this product. Issues with the IPOL vaccine include: 1. The clinical trials relied upon to license this product did not include a control group and only assessed safety for up to three days after injection, 2. IPOL allows for higher concentrations of vaccine antigens (8x for antigen type 3) in IPOL than were attainable in previous inactivated polio vaccines, enhancing the risk of IgG4 autoimmune disease (Kiszel et al, 2023), and 3. unlike the original inactivated vaccine, the virus used in IPOL is grown in Vero cells, a continuous line of monkey kidney cells cultivated on microcarriers. Vero cells have modified chromosomes which cause them to replicate forever, like cancer cells. These cells are susceptible to infection by dozens of viruses, including HPV, measles, rubella, reovirus, SV40 virus, and SV5. Vero cells do not secret the signal peptide interferon upon infection with viruses and therefore the anti-viral defense mechanism of the cell is impaired (Emeny and Morgan, 1979). Thus, the vaccines may be contaminated with antigens from other viruses or with viruses that have not been demonstrated to be inactivated.

The 1976 Swine Flu Vaccine Incident

The consequences of the Cutter polio vaccine is a regulatory landscape in which vaccines undergo thousands of tests to better ensure their safety and effectiveness. Yet this vital evidence-based process of vaccine development and testing for safety and efficacy has still been ignored. In 1976, concerns about the emergence of a new swine flu strain reminiscent of the lethal 1918 version led President Ford to convene a panel that recommended a government-backed mass vaccination program. Poorly conceived, the attempt to vaccinate the US population at breakneck speed failed in almost every respect (Trogen et al, 2020). Safety standards deteriorated and one manufacturer produced the incorrect strain. The vaccine tested poorly on children who, depending on the form of vaccine tested, either developed adverse reactions with high fevers and sore arms or did not mount an immune response. Reports emerged that the vaccine appeared to kill people and cause Guillain-Barré syndrome in a small number of cases, a finding that remains controversial, but added to the early momentum of the antivaccine movement (Spencer and Millar, 2006). Not having learned from the Cutter Incident, the eagerness in 1976 to rapidly distribute a vaccine undermined the scientific integrity of the process and damaged public trust.

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Table 1: Examples of viruses that can be propagated in Vero cells (from Kiesslich and Kamen, 2020)

Group	Family	Genus	Species	Envelope	Reference
I: dsDNA	*Adenoviridae*	*Mastadenovirus*	*Human adenovirus (HAdV)*	No	(Rhim et al., 1969)
	Herpesviridae	*Simplexvirus*	*Herpes simplex virus (HSV)*	Yes	(Rhim et al., 1969)
	Herpesviridae	*Varicellovirus*	*Varicella zoster virus (VZV)*	Yes	(Rhim et al., 1969)
	Poxviridae	*Orthopoxvirus*	*Vaccinia virus (VACV)*	Yes	(Rhim et al., 1969)
	Poxviridae	*Capripoxvirus*	*Sheeppox virus (SPV)*	Yes	(Trabelsi et al., 2014)
III: dsRNA	*Reoviridae*	*Orthoreovirus*	*Reovirus*	No	(Berry et al., 1999)
	Reoviridae	*Rotavirus*	*Rotavirus (RV)*	No	(Wu et al., 2017)
IV: +ssRNA	*Coronaviridae*	*Betacoronavirus*	*Middle East respiratory syndrome-related coronavirus (MERS-CoV)*	Yes	(Chan et al., 2013)
	Coronaviridae	*Betacoronavirus*	*Severe acute respiratory syndrome coronaviruses (SARS-Cov and SARS-Cov-2)*	Yes	(Ma et al., 2020; Spruth et al., 2006)
	Flaviviridae	*Flavivirus*	*Dengue virus (DENV)*	Yes	(Liu et al., 2008)
	Flaviviridae	*Flavivirus*	*Japanese encephalitis virus (JEV)*	Yes	(Wu and Huang, 2000)
	Flaviviridae	*Flavivirus*	*Yellow fever virus (YFV)*	Yes	(Souza et al., 2009)
	Flaviviridae	*Flavivirus*	*West Nile virus (WNV)*	Yes	(Lim et al., 2008)
	Flaviviridae	*Flavivirus*	*Zika virus (ZIKV)*	Yes	(Nikolay et al., 2018)
	Matonaviridae	*Rubivirus*	*Rubella virus (RuV)*	Yes	(Aubrit et al., 2015)
	Picornaviridae	*Enterovirus*	*Enterovirus A (EV-A71)*	No	(Wu et al., 2004)
	Picornaviridae	*Enterovirus*	*Enterovirus C/Poliovirus*	No	(Rhim et al., 1969)
	Picornaviridae	*Hepatovirus*	*Hepatovirus A (HAV)*	No	(Sun et al., 2004)
	Togaviridae	*Alphavirus*	*Chikungunya virus (CHIKV)*	Yes	(Tiwari et al., 2009)
	Togaviridae	*Alphavirus*	*Ross River virus (RRV)*	Yes	(Kistner et al., 2007)
V: -ssRNA	*Hantaviridae*	*Orthohantavirus*	*Hantaan orthohantavirus (HTNV)*	Yes	(Choi et al., 2003)
	Orthomyxoviridae	*Alphainfluenzavirus*	*Influenza virus A*	Yes	(Kistner et al., 1998)
	Orthomyxoviridae	*Betainfluenzavirus*	*Influenza virus B*	Yes	(Kistner et al., 1998)
	Paramyxoviridae	*Morbillivirus*	*Measles morbillivirus (MeV)*	Yes	(Rhim et al., 1969)
	Paramyxoviridae	*Morbillivirus*	*Peste des Petits ruminants virus (PPR)*	Yes	(Silva et al., 2008)
	Paramyxoviridae	*Orthoavulavirus*	*newcastle disease virus (NDV)*	Yes	(Rhim et al., 1969)
	Paramyxoviridae	*Orthorubulavirus*	*Mumps orthorubulavirus (MuV)*	Yes	(Rhim et al., 1969)
	Pneumoviridae	*Orthopneumovirus*	*Respiratory syncytial virus (RSV)*	Yes	(Rhim et al., 1969)
	Rhabdoviridae	*Lyssavirus*	*Rabies virus*	Yes	(Mendonca et al., 1993)
	Rhabdoviridae	*Vesiculovirus*	*Vesicular stomatitis virus (VSV)*	Yes	(Rhim et al., 1969)

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As Brownlee et al (2017) have written, "Overuse, which is defined as the provision of medical services that are more likely to cause harm than good, is a pervasive problem." The 1976 panel for swine flu was led by physicians (Sabin, Salk, Cooper, Spencer), not scientists. The race to vaccinate rather than use other means to prevent infections and outbreaks reflects the control of all our health institutes by physicians, not scientists, leading to calling those who scientifically question vaccines as "anti-vaxxers" (Cheng, 2022). To the extreme, when vaccines are mandated and people lose their jobs if they refuse to be vaccinated, Bardosh et al (2022) writing in the BMJ, "Our analysis strongly suggests that mandatory COVID-19 vaccine policies have had damaging effects on public trust, vaccine confidence, political polarization, human rights, inequities and social wellbeing. We question the effectiveness and consequences of coercive vaccination policy." The point is, we need rational, scientifically-based public health policies and therefore need scientists, not physicians, most of whom are not scientists (Smith, 2004), at the forefront of leading such policy decisions. Scientists understand the complexity of vaccines, physicians do not.

RSV Vaccines Failures

As stated in a FDA Briefing Document of Dec. 12, 2024 concerning Vaccine-Associated Enhanced Respiratory Disease (VAERD) in infants, "The mechanisms responsible for FI-RSV VAERD are still not fully understood; however, immune responses considered to contribute to the immunopathogenesis of VAERD" (https://www.fda.gov/media/184301/download). Further from the FDA Briefing, " In the mid-1960s, multiple published reports described an association between FI-RSV vaccines and VAERD (Fulginiti, 1969; Kapikian, 1969; Kim, 1969). In one study of a FI-RSV vaccine in infants, 80% of vaccine recipients required hospitalization for severe RSV-LRTD upon natural RSV infection, including two children who died at the ages of 14 months and 16 months of age; in comparison, no deaths occurred and 5% of participants required hospitalization in the control group (Kim, et al., 1969)." Now in 2024, from the same FDA report concerning vaccinating infants, "In July 2024, FDA was notified of a study pause in Phase 1 study mRNA-1365-P101 due to a study pause criterion being met. A potential safety signal for RSV sLRTI was identified, and as additional information accrued, an imbalance in cases of RSV sLRTI was noted, with more cases identified in the vaccine groups compared with the control group. This raised a concern for possible VAERD." In other words, in the 50+ years hence injuring infants with RSV vaccines and not understanding why, the FDA is allowing vaccines to be administered to infants without an understanding of how they work.

Covid-19 Vaccine Failures

Because Covid-19 vaccines have received so much attention and have been the subject of numerous studies, I'll highlight what's known about Covid-19 vaccine injuries. Vaccination programs for Covid-19 have been extensive globally, but most of these vaccines have been approved without extensive studies on their side-effects and efficacy (Chen et al, 2021). In general, adverse events associated with SARS-CoV-2 vaccination may be specific or non-specific. Specific adverse events may manifest themselves in the central and peripheral nervous system (CNS/PNS), heart, intestines, blood coagulation system, lymphatic system, bone marrow, or skin. From numerous published studies reviewed by Finsterer and Hertz (2024), the most common CNS complications to SARS-CoV-2 vaccination include sinus venous thrombosis, headache, acute disseminated encephalomyelitis (ADEM), acute encephalitis, and transverse myelitis. PNS complications following SARS-CoV-2 vaccinations include Guillain-Barre syndrome (GBS). Cardiac complications of SARS-CoV-2 vaccinations include acute myocardial damage, myocarditis or perimyocarditis. Gastro-intestinal side effects of SARS-CoV-2 vaccination include autoimmune hepatitis, nausea, and vomiting. There is a report of a fatal pulmonary embolism one day after the first dose of the AstraZeneca vaccine. The lymphatic system can react with lymphadenopathy. Bone marrow problems can manifest as hemolytic anemia or immune thrombocytopenia. Dermatological manifestations following SARS-CoV-2 vaccination include bullous rash, erythema, zoster, angioedema, wheals, scaly plaques, erythematous patches, and macules and papules. Oral and skin pemphigus have been reported (Pham et al, 2023; Nunez et al, 2024). Non-specific side effects include fever, fatigue, arthralgia, swelling, chills, warmth, myalgia and local injection site reactions including induration, tenderness and itching.

Demyelination is an underlying mechanism of autism (Galvez-Contreras et al, 2020). Although some epidemiological studies have not found an association between vaccination and MS or demyelination (Hapfelmeier et al, 2019), the incidence of Covid-19 vaccines causing demyelination has been reported (Paybast et al, 2024). Other autoimmune conditions, including vitiligo, have been reported in studies of a large number of people following Covid-19 vaccination (Kim et al, 2024). Evidence suggests a pathogenic role of cutaneous dysfunction, such as vitiligo, in ASD (Man et al, 2023). The connection between skin and brain begins in the organ's origins in the neuroectoderm (Zoccante et al, 2022). 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

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inducing autoimmune disease needs to be studied (Chen et al, 2001; Toussirot and Bereau, 2015; Principi and Esposito, 2020; Chen et al, 2022).

Vaccine Induced Autoimmunity

Molecular mimicry refers to a significant similarity between certain pathogenic elements contained in the vaccine or made by the vaccine, such as the proteins made by the mRNA vaccines, and specific proteins native to the human host. The similarity of the two may lead to immune cross reactivity, creating a reaction of the immune system towards the pathogenic antigens may harm the similar human proteins, thus causing autoimmune disease (Segal and Shoenfeld, 2018). Immune responses vary greatly among individuals. For example, cytokine responses induced by Bacille Calmette-Guerin (BCG) vaccination show~100-fold variability (Querec et al, 2009). Seven cases of de novo Rheumatoid arthritis following COVID-19 vaccination have been reported, with a higher incidence in women (4/7, 57%). Four of these were reported after the mRNA vaccine and one after the Viral vector vaccine (Guo et al, 2023). Maternal rheumatoid arthritis is a known risk factor for ASD in their offspring (Yin et al, 2023).

Possible Mechanisms of Vaccine Injury

IgG4

Vaccines induce an overresponse in IgG4, and children with autism have increased IgG4 (Croonenberghs et al, 2002; Einstrom et al, 2009; Zaky et al, 2018). Espino et al (2024) found a class switch toward the antibody isotype IgG4 was induced a few weeks after the third dose of mRNA Covid-19 vaccine, which peaked abruptly and remained at high levels for a long period, a condition found in many autoimmune diseases (Huijbers et al, 2018). Along with an IgG4 response, Portilho et al (2024) found an unexpected IgE-mediated immune response to SARS-CoV-2 vaccination, another mechanism underlying autoimmune disease (Maurer et al, 2018) that is typically triggered by allergens. The propensity of mRNA vaccination to induce IgG4 antibodies, like many aspects of vaccination, was not predicted (Pillai, 2023). As Pillai writes, "on the basis of the results of the studies discussed here and other theoretical considerations, future clinical studies need to evaluate the effectiveness of temporal spreading out of mRNA vaccine boosts—possibly no more than once a year. Other approaches worth investigating would be the use of smaller quantities of mRNA for booster doses and, separately, the use of mRNA vaccines for priming only, with heterologous boosts with adjuvant-free recombinant spike proteins because, theoretically, adjuvants are most relevant during priming and may not be necessary for boosting. Hybrid immunity, as generated by breakthrough infections after vaccination, can also induce anti-spike IgG4, so there is a need for ongoing evaluation and possibly tweaking of mRNA vaccination strategies going forward."

Recent data from Kiszel et al (2023) provide evidence that IgG4 is a major mediator of mRNA vaccine-induced autoimmune disease. The most abundant antibody (also called immunoglobulin) isotype in the human serum (blood is different from mucosa where IgA dominates) is immunoglobulin G (IgG). The subclasses of IgG are very similar but differ in their constant regions (the region of antibody used to destroy antigens). Each subclass has a unique profile in terms of antigen binding, immune complex formation, complement activation and triggering of effector cell activation. After antigenic stimuli, IgG3 and IgG1, the two main complement-activating subclasses are secreted first, whereas IgG2 and IgG4, which are formed later, are thought to play a role in attenuating inflammation due to their inability to activate complement. Previous studies have found that antibody responses to viral protein antigens are mainly restricted to IgG1 and IgG3. IgG2 is stimulated primarily by carbohydrate antigens, whereas IgG4 is produced in response to prolonged antigen stimulations. mRNA vaccination yields a higher antibody titer than does the SARS-CoV-2 infection. In other words, a huge amount of antigen presentation elicited by the mRNA vaccination induces a huge amount of antibody production – too much. That huge antibody production means a high level of autoantibodies, such as IgG4.

Goldman and Miller (2023) found a positive correlation between the number of vaccine doses and infant mortality rates is detectable in the most highly developed nations but attenuated in the background noise of nations with heterogeneous socioeconomic variables that contribute to high rates of infant mortality, such as malnutrition, poverty, and substandard health care. In an analysis of multiple vaccinations, Mawson and Croft (2020) found that multiple vaccination is linked to neurological developmental disorders (NDD) and that preterm birth combined with vaccination was associated with a synergistically 6.6-fold increased odds of NDDs. The authors provide evidence for a hypothesis that acute injury and chronic multisystem illness related to vaccines involve liver dysfunction and the release of stored vitamin A compounds into the circulation in toxic concentrations, leading to endogenous forms of hypervitaminosis A. They also advise, "Further studies of health outcomes in vaccinated and unvaccinated groups are urgently needed: to increase understanding of the pathophysiology and treatment of vaccine injury; to identify the risk factors and to screen for vaccine injury; to inform public health policy on potential hazards related to vaccination schedules; and to optimize the safety and benefits of vaccines."

Considering Covid-19 (Boreti, 2024), up to a certain level, IgG4 provide a protective effect. Above that level, IgG 4 makes the immune system excessively susceptible

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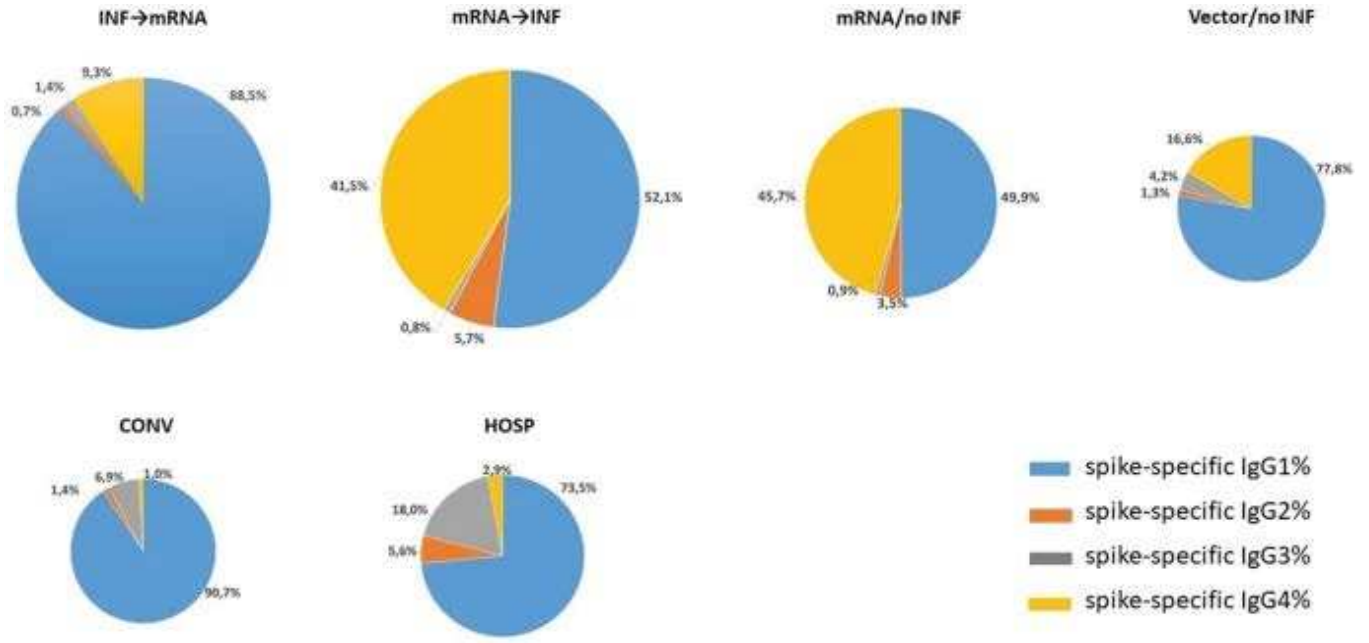
to the COVID-19 spike protein. Higher levels of IgG 4 were previously associated with patients who perished of COVID-19 infection. According to Uversky et al (2023), increased IgG4 synthesis due to repeated mRNA vaccine boosters may cause autoimmune diseases, and promote cancer growth and autoimmune myocarditis in susceptible individuals. IgG4 can mediate autoimmune diseases and create gut dysbiosis and leaky gut (Wang et al, 2018), potentially leading to a number of maladies, including neurological disorders and ASD (Fowlie et al, 2018). However, IgG subclasses produced against protein antigens depend on factors other than the type of pathogens or type of vaccine, such as T-helper cell response, and the route and the site of infections or injections. As such, the intricacies of how an individual will respond to a particular vaccine is largely unknown. The drug companies don't want to know because their business model is to give a drug to as many people as possible, and knowing about injuries is an impediment to making money. Leave it to academic researchers who aren't paid-off by pharma to figure out the safety data. Alas, there is not enough money in academia to do this well, but Kiszel et al (2023) give us important info.

Let's look at what mRNA vaccines do to the different antibody levels. Here's Figure 5 from Kiszel et al (2023).

Notice that the percentages of spike-specific IgG4 were



higher in the vaccinated groups than in the COVID-19 infected groups. The proportions of the spike-specific IgG4 subclass to the sum of all spike-specific IgG antibodies were between 1 and 3% in the infected groups. However, in the vaccinated groups, they detected 16.6% of spike-specific IgG4 in the Vector/no INF group, whereas its values were as high as 41.5% and 45.7% in the mRNA→INF and mRNA/no INF groups, respectively. That is, those who were mRNA-vaccinated but had no previous Covid-19 infection (mRNA –>INF) or who had no Covid-19 infection before or after vaccination (no INF), had high levels of IgG4.

The consequences of IgG4 autoantibodies in the CNS are many, including hypertrophic pachymeningitis characterised by localised or diffuse inflammation and thickening of the meninges (mostly dura mater)—cerebral, spinal or rarely both and the cranial nerves (Baptista et al, 2017). The effects on the cranial nerve may manifest in tinnitus, something frequently afflicting those with autism (Timms et al, 2022).

Koneczny et al (2022) have pointed out that IgG4 autoimmune diseases (IgG4-AID) appear to be distinct from another group of rare immune diseases associated with IgG4 that are known as the IgG4-related diseases (IgG4-RLD). Repeated vaccinations cause long-term elevation of IgG4 (Buhre et al, 2023). In IgG4-RLD, an increased serum concentrations of IgG4 is present in a majority of patients, but the antigenic targets of IgG4 and the role of IgG4 antibodies for pathogenicity has not been elucidated. In contrast, IgG4-AID patients do not have the 10-fold increase in serum IgG4 levels as IgG4-RLD patients, but are associated with antigen-specific, pathogenic IgG4 autoantibodies that are known to target the central and peripheral nervous system.

Key here is that antibodies have the highly variable regions of heavy and light chains that are known as Complementarity-Determining Regions (CDRs). CDRs

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confer target binding high-affinity and specificity, generated through clonal selection of immune B-cells that have produced antibodies with varying affinities by both V(D)J recombination and somatic hypermutation of hypervariable DNA regions corresponding to CDRs (MacCallum et al (1996). When antigens are encountered, the naive B cells undergo somatic hypermutation (SHM)18 in the V exons and affinity-based selection in the germinal centers (Di Noia and Neuberger, 2007). Importantly, the more antigen presented to B-cells, such as in vaccinations that produce large amounts of antigen and repeated vaccines that produce large and sustained amounts of antigen, the more B-cell expansion occurs (Donahue and Fruman, 2003). This antibody somatic hypermutation can potentially lead to autoantibodies that attack self because the random mutations introduced during this process may create antibodies that binds to a self-antigen (Elkorn and Casali, 2008). Each B-cell may potentially make 2 or more antibodies or autoantibodies types (Shi et al, 2019).

B- cell hypermutation occurs after vaccination (Jacob-Dolan, 2024), which can then lead to the production of autoantibodies (Deguine and Xavier, 2024). Many studies have demonstrated that failure of tolerance occurs at multiple independent checkpoints leading to autoantibody production in different antibody-producing pathways, particularly during B cell maturation, in blood circulating plasmablasts during differentiation, and within germinal centers. These checkpoint failures are marked by distinct repertoire features that may be used to identify disease- or patient-specific therapeutic approaches, including those associated with Autism. Individuals with autism spectrum disorder (ASD) can exhibit a variety of autoantibodies types, thus producing antibodies that mistakenly target multiple sites in their own body tissues, particularly brain proteins (Zhou et al, 2020). Indeed, multiple autoantibody types have been found in maternal blood that are associated with that mother's child having ASD (Ramirez-Celis et al, 2022).

Treg Suppression and T-cell Antigenic Mimicry

"Suppression of Treg cells by adjuvants may constitute an ideal vaccination strategy for generation of protective antiviral T cell immunity" (Lin et al, 2018). Adjuvants can enhance the immunogenicity of T cell vaccines through different mechanisms, including restricting the development of Treg cells, and promoting the T cell immunity (Bayry, 2014). Regulatory T cells (Tregs) play a critical role in regulating tissue inflammation, and reduced Treg numbers and/or suppressive function contribute to autoimmune disease (Gosswami et al, 2022). Further, antigenic mimicry can lead to autoimmune diseases in which T cells don't distinguish environmental antigens from self proteins (Wildner, 2023). Previous studies have found acquired autoimmunity after viral vaccination that is caused by molecular mimicry and antigen complimentarity (Root-Bernstein, 2007) in the presence of an immunologic adjuvant (Waisbren, 2008).

Persistent antigen stimulation, as in vaccines and chronic infections, results in T cell exhaustion (TEX) —a state of impaired effector functions. TEX cells shifted from acetate to citrate metabolism by downregulating acetyl-CoA synthetase 2 (ACSS2) while maintaining ATP-citrate lyase (ACLY) activity. This metabolic switch increased citrate-dependent histone acetylation, mediated by histone acetyltransferase KAT2A-ACLY interactions, at TEX signature-genes while reducing acetate-dependent histone acetylation, dependent on p300-ACSS2 complexes, at effector and memory T cell genes. Nuclear ACSS2 overexpression or ACLY inhibition prevented TEX differentiation and enhanced tumor-specific T cell responses (Ma et al, 2024). Eating fiber leads to the production of acetate in the gut, a short chain fatty acid that helps to prevent T-cell exhaustion.

As reviewed by Terrabuio et al (2023), in acute inflammatory responses, when antigen is effectively cleared, short-lived effector T cells undergo controlled apoptosis, while long-lived effector T lymphocytes differentiate into memory T cells, thus efficiently resolving the inflammatory reaction. However, during chronic inflammatory conditions, such as in hyper inflammatory syndrome following vaccination (Ouldali et al, 2022), this natural resolution is impaired, and CD8+ T lymphocytes become exhausted or senescent, retaining a neurotoxic potential and contributing to several neurodegenerative diseases. CD8+ T cells, reacting against self and non-self antigens are clonally expanded in many brain disorders. Although these disorders may have distinct causes, occurrence rates, and clinical presentations, they share common immunopathological characteristics. These include the circulating origin of CNS-invading CD8+ T lymphocytes, the clonal expansion of CD8+ T cells, and phenotypical traits that resemble senescence. A single vaccine boost of a mRNA vaccine for cancer has been found to markedly extend T-cell clone lifespan sevenfold to a median lifespan of 7.7 years, with lifespans ranging from 1.5 to 100 years (Sethna et al, 2025). In other words, the autoimmune potential of T-cell clones induced by a vaccine may last for 100 years with a median lifespan of 7.7 years.

Vaccination and Infection Induce Gut Dysbiosis

In a study of 34 subjects who had stool collection prior to vaccination and one month post vaccination to evaluate the relative abundance of *bifidobacteria* in the gut, the relative abundance of genus *bifidobacteria* was significantly decreased to about half of original value after vaccination (Hazan et al, 2022). Oral probiotics containing *bifidobacteria* have been reported to lessen the symptoms and mortality rate of Covid-19 infection (Bozkurt and Bilen, 2021). *Bifidobacterium* synthesize GABA, as the bacterium level

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decreases in autism, children with autism have low levels of GABA. This bacterium also has role in synthesis of Tryptophan and bile acid that acts as a precursor of Serotonin. Hence, lower levels of *bifidobacteria* leads to less serotonin in brain and can be correlated to autistic behavior (Mehra et al, 2023).

Vaccine Adjuvants

Alum (AlK(SO4)2), aluminum potassium sulfate, is an adjuvant commonly utilized in vaccines, and is a ubiquitous element to which contemporary life commonly exposes us. Food, air, water, waste, the earth's surface, and pharmaceuticals all represent means of aluminum (Al) exposure. As Lerner (2012) has pointed out, Al fits the diagnostic criteria of the newly described autoimmune/inflammatory syndrome induced by adjuvants, and warrants more study. Research data suggests that vaccines containing Al may be neurotoxic, transport to the brain (Mold et al, 2016) and be a contributing etiological factor in the increasing incidence of autism (Tomljenovic et al, 2014). While the source of Al could not be identified, Exley and Clarkson (2020) have found aluminium content of brain tissue in Alzheimer's disease, autism spectrum disorder and multiple sclerosis was significantly elevated compared to control brains. Some will argue that Al is eliminated from the body within days (Mitkus et al, 2011), but if true, why do some people have abnormally high levels of Al in their brains. Aluminum can cross the blood-brain barrier and accumulate in glial and neural cells (Wang, 2018) and increase oxidative stress (H2O2) in the hippocampus, diencephalon, cerebellum, and brain stem (Yuan et al, 2012).

In a study of infant monkeys, the authors (Burbacher et al, 2005) concluded that "knowledge of the biotransformation of thimerosal, the chemical identity of the Hg-containing species in the blood and brain, and the neurotoxic potential of intact thimerosal and its various biotransformation products, including ethylmercury, is urgently needed to afford a meaningful interpretation of the potential developmental effects of immunization with thimerosal-containing vaccines in newborns and infants. This information is critical if we are to respond to public concerns regarding the safety of childhood immunizations." Although the US banned the use of thimerosal in pediatric vaccines, it's still widely used in the US and many other jurisdictions. The CDC states that pediatric vaccines using thimerosal are safe and don't cause autism, but when you look at the study they cite (Price et al, 2010), it's from a private group (Abt Associates) that is funded by pharma companies and recently announced that "Abt Associates announced today the sale of its wholly-owned subsidiary Abt Bio-Pharma Solutions, Inc. (ABS) to United BioSource Corporation (UBC), a global scientific and medical affairs organization that partners with life science companies to develop and commercialize biopharmaceuticals, medical devices, and other health care technologies" (https://www.biospace.com/abt-bio-pharma-solutions-inc-acquired-by-united-biosource-corporation). Hardly an independent study.

Exposome Epigenetic Mechanisms

Epigenetics has been defined as "the study of changes in gene function that are mitotically and/or meiotically heritable and that do not entail a change in DNA sequence (Dupont et al., 2009). Following twins for life has provided evidence that early life can affect later life health through DNA methylation. These studies of twins and unrelated subjects have demonstrated that the sites relevant to the regulation of gene expression (CpG sites) with the strongest heritability are also those that are most influenced by the exposome (Li et al, 2022). Neurodevelopmental disorders such as autism are also emerging as having distinct epigenomic patterns (LaSalle, 2023). Further, epigenetic transgenerational inheritance is another means by which non-genomic, epigenetic changes can be passed to offspring. Epigenetic changes in blood samples two months following mRNA vaccination have been observed in humans (Pang et al, 2022), but more long term and transgenerational studies are needed.

Germline Disruption

Because autoantibodies can be carried in exosomes (Hua et al, 2024), the autoantibodies can be carried to the germline by the exosome where the autoantibody or other inflammatory signaling molecules may possibly alter the epigenetics of the germline (Wong et al, 2024). Actions of autoantibodies such as modification of post-translational modification of proteins involved in epigenetic regulation are possible (Lastwika and Lampe, 2024). More studies of these mechanisms are needed.

Protein Level Inheritance and Protein Aggregates

Epigenetics can refer to changes in phenotype that are not rooted in DNA sequence (Harvey et al, 2018). Epigenetics has largely been studied in the context of chromatin modification. However epigenetic traits can also be linked to self-perpetuating changes in proteins, individually or collectively. Many proteins that act as epigenetic elements, including prions, are intrinsically disordered or partially disordered. These disordered protein conformations do not adopt a single structure in the cellular milieu (Wu and Fuxreiter, 2016). Inflammation, associated with vaccination, can lead to an increases in disordered proteins (Lipton et al, 2007) and an increase in neurodegeneration (Zhang et al, 2023). Thus, protein-based inheritance is a mechanism by which intrinsically disordered proteins, possibly induced by vaccine related inflammation, can drive the emergence of new traits, adaptive opportunities, and possible neurodegeneration (Chakrabortee et al, 2016).

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Not only can a humoral immune response from vaccines induce protein aggregation (Ratanji et al, 2014), but the vaccines themselves many be delivering protein aggregates to the recipient (Agarwal et al, 2020). Protein aggregates can then induce physiological dysfunction and the aggregation of other protein types (Ajmal, 2023; Housmans et al, 2023), leading to possible neurodegenerative diseases, such as autism.

The One Size Fits All Problem

Predicting the degree to which an individual will mount an adequate immune response to vaccination and develop sustained immunity poses an ongoing challenge to determine vaccine safety and efficacy. What is altered in a newborn baby's immune system and what is altered in an older adult's immune system are not the same things. For example, in the elderly, a poor humoral immune response to influenza vaccination is attributed to immunosenescence. In a study in healthy 50–74-year-olds, although the global DNA methylation profile underwent minimal changes, a specific group of CpG sites, when co-ordinately hypomethylated, was associated with lower humoral immune response to influenza vaccination (Zimmerman et al, 2016). A study comparing influenza vaccine responses in older and younger populations found larger epigenetic remodeling in vaccine responders aged over 50 years (Gensous et al, 2018). In other words, a vaccine made to induce a satisfactory humoral response in older adults may induce an overresponse in younger adults and may lead to vaccine injury.

mRNA Vaccines Incorporate Into Genome and Resultant Spike Protein Lasts for Months

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, such as Paul Offit, who benefits by many millions of dollars from vaccines (Attkisson, 2008) having been a secondary author (see: https://patents.justia.com/inventor/paul-offit) on several vaccine patents developed by H. Fred Clark who has a Ph.D. in microbiology and immunology, in the media have said (Offit, 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). While we don't know whether the spike mRNA is inserted into our genomes, and, if so, whether that DNA would be expressed or suppressed, we do have evidence that spike proteins are expressed for at least two months following Covid-19 mRNA vaccination. A study published in May 2021 documented for the first time circulating vaccine-induced S protein in the blood of 11 out of 13 subjects as early as one day after injection of the Moderna COVID-19 vaccine, up to 150 pg/mL and for about two weeks after injection (Ogata et al, 2022). In another study, both vaccine mRNA and vaccine-induced S protein were found in lymph nodes up to 60 days after the second dose of either the Moderna or BioNTech–Pfizer COVID-19 vaccines (Roltgen et al, 2022), suggesting that endogenous production of S protein following vaccination may occur for much longer than many had thought.

New studies from scientists at Yale that haven't yet been peer-reviewed (Bhattacharjee et al, 2025), find that people with post-Covid-19-vaccination syndrome had reactivation of Epstein-Barr virus (Herzum et al, 2022) and significantly higher plasma levels of the coronavirus spike protein than other groups, including those with long Covid, from 26 to 709 days after receiving the vaccine. The mRNA vaccines are unlikely to be the source of the spike protein so long after the vaccines were administered, and, therefore, something else is allowing late-phase expression of spike protein, possibly incorporation of spike DNA through reverse transcription of the mRNA into DNA and incorporation of the spike protein DNA into the genome. Other possibilities exist too, such as a latent SARS-CoV-2 infection producing spike protein. Further research is imperative, immediately. Spike protein itself when systemically injected into mice can induce inflammation in the brain and increase levels of amyloid precursor protein, leading to an increase in misfolded proteins (Rong et al, 2024). Therefore, spike protein formation from vaccines may induce inflammation and protein misfolding in the CNS. Given that spike protein has been found in many tissues of those vaccinated with the mRNA vaccine (Cosentino and Marino, 2022), the authors suggest that "Taken as a whole, evidence strongly supports the possible link between inappropriate expression of S protein in sensitive tissues and subsequent tissue damage." In a follow-up analysis, Cosentino and Marino (2023) further state that, "healthy young people who are at minor risk of COVID-19 complications, COVID-19 vaccination-associated risks likely outweigh any possible expected benefits." Further, the spike protein contains a superantigenic motif known to elicit a hyperinflammatory adaptive immune response (Cheng et al, 2020). Evidence also finds that the spike protein drives NLRP3 inflammasome activation in human microglia (Albornoz et al, 2023), a possible mechanism in developing neurological symptoms following Covid-19 infection or vaccination. One explanation for this happening is that the virus, or vaccine related proteins, can now target vascular endothelial cells and disseminate to the CNS through a hematogenous mechanism. Once at the blood-brain-barrier (BBB), SARS-CoV-2 or vaccine related protein, binds the zonulin receptor and promotes zonulin release. Then zonulin,

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via PAR2, induces blood-brain-barrier (BBB) disruption allowing the virus or protein to enter. Disruption of barrier function in epithelial and endothelial cells has been found by UC Berkeley scientists to be mediated by the spike protein alone (Biering et al, 2022), meaning that the spike protein made by mRNA vaccines can mediate this disruption of barrier function.

Post-Vaccine Neurological Injury Reported by Prominent Physicians

Reported in Science, Covid19 vaccines can induce long-Covid-like symptoms (Couzin-Frankel and Vogel, 2022). 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).

Hertz et al (2023) recently reported her experience in a PubMed listed journal as:

"66yo previously healthy female who developed facial tingling, oral numbness, blurred vision, dizziness, and an elevated blood pressure of 176/127 mmHg, 30 min after receiving the first dose of the BNT162b2 vaccine (Biontech Pfizer Vaccine [BPV]) with the lot (batch) number EK5730 in 12/2020. One day later, she developed severe dysesthesias of the entire integument ("burning skin"), predominantly of the face and head, as well as diffuse numbness, tingling, twitching, electrical feelings throughout, internal vibrations, tinnitus, blurred vision, tremors, trouble speaking, profound weakness, post-exertional malaise and brain fog. She also experienced a tight band-like constriction around the chest, left-sided chest pain, pounding tachycardia, and shortness of breath. She noted watery diarrhea for the first two weeks followed by severe constipation. She also experienced dysautonomia manifested by dizziness, imbalance, Postural Tachycardia Syndrome (POTS), urinary hesitancy, and profuse sweating. Heart rate increased by 25–30 points when standing. She also noted excessive hair loss. Anti-ACE-2 antibodies were elevated at 13.7 (n, < 9.8 U/mL)." In another study by Krumholz et al (2023) at Yale School of Medicine, 241 individuals who reported post-vaccine syndrome (PVS) after covid-19 vaccination had low health status, high symptom burden, and high psychosocial stress despite trying many standard medical treatments. The authors call for continued investigation to understand and treat this PVS condition. As Avindra Nath (2023) at the NIH has published, "Action is needed that brings together manufactures, healthcare agencies, clinical and bench scientists, and legislatures on a global platform to investigate vaccine-related neurologic adverse events and develop ways to prevent and treat them."

Vaccine-Induced Rapid Evolution Through Genotypic, Epigenetic, and Phenotypic Change

Whether the adaptive immune system is activated by a pathogen or a vaccine in lieu of that pathogen, the rapid replication of B-cells with resultant mutations and production of many antibody types results. The more antigen presented, the more rapidly B-cells proliferate and mutate (Gitlin et al, 2014). These high rates of B cell proliferation lead to increased rates antibody production and mutation that can protect from pathogens but also promote autoimmunity. Thus, B cells experience extreme alterations in their metabolism throughout their life cycle, from naïve B cells, having minimal activity, to germinal center (GC) B cells that proliferate at the fastest rate of all cells, to long-lived plasma cells that express very high levels of protein production that can persist for decades. The mechanisms responsible for these B-cell transitions remain incompletely understood (Johnstone et al, 2024). Metabolic control of B cell function is lost in autoimmunity (Raza and Clarke, 2021) and high lactate levels in many ASD patients (Sharpe et al, 2013) may be one indication that when ASD patients were vaccinated, metabolic control of their B-cells was not well controlled and led to autoantibody production and production of inflammatory cytokines (Romero et al, 2024).

For example, a 6-month longitudinal study of individuals who never had SARS-CoV-2 infection compared with people who had recovered from SARS-CoV-2, found that the frequency of SARS-CoV-2–specific memory B cells continued to increase from 3 to 6 months postvaccination. mRNA vaccines also generated a higher frequency of variant cross-binding memory B cells than mild SARS-CoV-2 infection alone, with >50% of RBD-specific memory B cells cross-binding all three variants of concern at 6 months (Goel et al, 2021). The variant-binding memory B cells were more hypermutated than wild-type–only binding cells. Even

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rare bnAb-precursor naive B cells with well-defined genetic signatures were activated by immunogens and subsequently matured to bnAb development (de Camp et al, 2024), leading to more mutations. Environmentally regulated epigenetics will also induce variability in the B cell production of antibodies (Moroney et al, 2020), where, for example, SCFAs derived from fermentation of dietary fiber may limit autoantibody production through inhibition of histone deacetylation (Sanchez et al, 2020). The hypermutation of B cells or their precursors and the resulting antibodies that they make means that each individual will rapidly evolve new genotypes, epigenotypes, and phenotypes in their adaptive immune system. Hence, individual variation, including variation in autoantibody production will occur in individuals, meaning individuals will respond differently to a given vaccine. Vaccine injury may occur in some, but not others as a result.

Vaccine-Induced Cancer and Correlation with Autism

Axillary and cervical lymphadenopathy following vaccination is a well-known for smallpox, influenza, human papilloma virus and other vaccines. Recently, COVID-19 vaccination was identified as a cause of inflammatory unilateral axillary and cervical adenopathy. The median duration of adenopathy reported after mRna-1273 (Moderna) and BNT162b2 (Pfizer) vaccine administration ranges from 1–2 days to approximately 10 days in duration, but in some cases can persist for more than 1 month after vaccination (Cavanna et al, 2023) – now these authors report that Non-Hodgkin Lymphoma developed shortly after mRNA COVID-19 vaccination. Others report similar results (Sekizawa et al, 2022). There is an increased cancer risk among individuals with autism, which may be exacerbated by co-occurring intellectual disability and/or birth defects in ASD (Liu et al, 2022). High rates of b cell proliferation combined with inflammation following vaccination may present a state conducive to cancer, autoimmunity and autism, something for which a strong correlation exists (Kao et al, 2010; Chiang and Teng, 2014).

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 8thcranial nerve, leading to vestibular dysfunction (Girasoli et al, 2018). In vaccine injury cases, symptoms of tinnitus, vestibular dysfunction such as dizziness, vertigo, and loss of balance, along with nystagmus and therefore degraded visual acuity can be present. The constellation of symptoms suggest 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). Long-term blistering (pemphigus) of the gums and mouth cavity has occurred for many months in some patients. 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).

Prevention and Remediation Strategies

I've previously published about means to better prevent being infected by boosting immunity through lifestyle (Maguire, 2020), how to produce a non-classical vaccine without antigenic presentation by boosting the immune system with bioidentical molecules released from stem cells (Maguire, 2021), how to better make vaccines so that over-antigen production and vaccine injury are reduced (Maguire, 2022), and how to use diet and lifestyle strategies to mitigate the autoimmune effects of vaccine-induce autoimmune disease (Maguire, 2023).

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). Omnivore diets are associated with bacterial

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species such as *A. putredinis, B. wadsworthia* and *R. torques*, that were linked to meat (especially red versus white meat) consumption. These species have been previously implicated in inflammatory diseases and an overall decrease in SCFAs (Fackelman et al, 2025). Evidence about the dietary interventions for the autoimmune disease, Multiple Sclerosis, are instructive. Stoiloudis et al (2022) have reviewed the reported adverse effects of saturated fatty acids (SFAs) on the course of MS, emphasizing their proinflammatory character. High intake of SFAs leads to a dysbiosis of gut microbiota. Additionally, the consumption of vegetable oils, which are enriched with trans fatty acids, is associated with gut inflammation and the upregulation of proinflammatory cell. Red meat leads to the formation of nitrous compounds increasing chronic inflammation. Red meat also contains arachidonic acid, which participates in inflammatory pathways by activating Th17 cells. Furthermore, a high consumption of sugar-sweetened beverages and refined cereals leads to the production of insulin, which, in this way, is responsible for the upregulation of synthesis and the production of arachidonic acid. High salt intake can induce the production of Th17 cells and proinflammatory cytokines. Proteins contained in cow-milk may play a role in the mechanisms of pathogenesis of MS. Particularly, butyrophilin can induce EAE by mechanisms of molecular mimicry with myelin oligodendrocyte glycoprotein.

Clinical studies suggest that dietary modifications, particularly the elimination of dairy, can decrease in the blood of Folate Receptor Antibody (FRA), an autoantibody that impedes the high affinity folate receptor (FRα), hindering the transfer of folate across the blood-brain barrier. This reduction in FRA levels may restore the brain's uptake of folate. Recent studies have found that 70% of ASD (autism spectrum disorder) children exhibit FRA (Ayoub, 2023). Recent studies have also found that folate may be involved in the production and/or differentiation of endogenous neural stem cells neural stem cells (Mohanty et al, 2017). FRA would then likely impede neural stem cell activity potentially leading to neural deficits. Unfortunately, vaccines against FRα are currently in clinical trials for cancer (Yeku et al, 2018), the side effects on the brain, including mothers and their fetuses, are not understood. 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, low in sodium, 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 glucose, because it disrupts gut barrier function (Zhang et al, 2021), and 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. 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

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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. A combination of amino acids, prebiotics, probiotics, and postbiotics has been proposed to rebuild the gut barrier function and rescue the degenerating microvilli (Maguire and Maguire, 2019).

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). Low selenium levels (McLachlan et al, 2017), and high iodine levels (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). Signature microbes of the gut microbiome in vegans, included *Lachnospiraceae, Butyricicoccus sp.* and *R. hominis*, linked to the consumption of fruits and vegetables and having a specialized role in fiber degradation, and are producers of SCFAs (Fackelmann et al, 2025). 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. Eating fiber leads to the production of acetate in the gut, a short chain fatty acid that helps to prevent T-cell exhaustion. 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 autoimmunological disorders. Polyphenols are capable of activating intracellular pathways (e.g., the arachidonic acid-dependent pathway, the nuclear transcription factor (NF-κB), 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).

Conclusions

The geo-epidemiological distribution of ADs, including ASD, 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 predominantly driving these evolutionary processes (Mazzuca et al, 2021). Indeed, the exposome underlies most diseases (Rapapport and Smith, 2010). Part of the mechanisms underlying the increase in ADs and ASD 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) and the vaccines themselves, and the effects gut permeability may have on vaccine safety, and variability of efficacy and safety in individuals (Hong, 2023). Rubio-Cassilas et al (2024) have proposed that the synthetic spike protein produced by the Covid-19 mRNA vaccine can enter the intestinal cells and trigger an inflammatory response, thus affecting the delicate balance between the gut microbiota and intestinal cells. The proposed sequalae following vaccination may include gut dysbiosis as seen in other species (Wu et al, 2022) and leaky gut syndrome. Vaccine induced autoimmune disease, and ASD needs to be studied at multiple levels, including epidemiologically to understand it 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. As Platschek and Boege (2024) at the Heinrich Heine University, University Hospital, in Düsseldorf, Germany, have written, "Shortcomings of pharmacovigilance, lack of corresponding reactions by national health authorities and unvalidated changes of vaccine composition may have contributed to overlooking PACVS [post-acute-Covid-vaccination syndrome." While this statement is specific to Covid vaccines, the statement is true for vaccines in general. We need to study the long term consequences of vaccines and use these data to make our vaccines more efficacious and safer.

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