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PREMIUM **HEALTH NEWS**

SARS-COV-2 Vaccines and Neurodegenerative Disease

By Stephanie Seneff and GreenMedInfo | January 11, 2022 Updated: January 12, 2022 Print

Since December 2020, when several novel unprecedented vaccines against SARS-CoV-2 began to be approved for emergency use, there has been a worldwide effort to get these vaccines into the arms of as many people as possible as fast as possible. These vaccines have been developed "at warp speed," given the urgency of the situation with the COVID-19 pandemic. Most governments have embraced the notion that these vaccines are the only path towards resolution of this pandemic, which is crippling the economies of many countries.

use for protection against COVID-19 in the US and/or Europe. Two (the Moderna vaccine and the Pfizer/BioNTech vaccine) are based on mRNA technology, whereas the other two (produced by Johnson & Johnson and AstraZeneca) are based on a double-stranded DNA recombinant viral vector. The mRNA vaccines contain only the code for the SARS-CoV-2 envelope spike protein, whereas the DNA-based vaccines both contain an adenovirus viral vector that has been augmented with DNA that codes for the SARS-CoV-2 spike protein. The DNA-based vaccines have a certain advantage over the RNA-based vaccines in that they do not have to be stored at deep-freeze temperatures, because double-stranded DNA is much more stable than single-stranded RNA. But a disadvantage is that those who have been exposed to natural forms of the adenovirus have antibodies to the virus that will likely block the synthesis of the spike protein, and therefore not afford protection against SARS-CoV-2.

In this regard, the AstraZeneca (AZ) vaccine has a slight advantage over the Johnson & Johnson (J&J) vaccine because the virus normally infects chimpanzees rather than humans, so fewer people are likely to have been exposed to it. On the other hand, several studies have shown that viruses that normally infect one species can cause tumors if they are injected into a different species. For example, a human adenovirus injected into baboons caused retinoblastoma (cancer of the eye) in the baboons . So, it can't be ruled out that the AZ vaccine could lead to cancer.

People don't realize that these vaccines are vastly different from the many childhood vaccines we are now used to getting early in life. I find it shocking that the vaccine developers and the government officials across the globe are wrecklessly pushing these vaccines on an unsuspecting population. Together with Dr. Greg Nigh, I recently published a peer-reviewed paper on the technology behind the mRNA vaccines and the many potentially unknown consequences to health . Such unprecedented vaccines normally take twelve years to develop, with only a 2% success rate, but these vaccines were developed and brought to market in less than a year. As a consequence, we have no direct knowledge of any effects that the vaccines might have on our health over the long term. However, knowledge about how these vaccines work, how the immune system works and how neurodegenerative diseases come about can be brought to bear on the problem in order to predict potential devastating future consequences of the vaccines.

the SARS-CoV-2 virus. However, both the mRNA and the protein it produces have been changed from the original version in the virus with the intent to increase rate of production of the protein in an infected cell and the durability of both the mRNA and the spike protein it codes for. Additional ingredients like cationic lipids and polyethylene glycol are also toxic with unknown consequences. The vaccines were approved for emergency use based on grossly inadequate studies to evaluate safety and effectiveness.

Our paper showed that there are several mechanisms by which these vaccines could lead to severe disease, including autoimmune disease, neurodegenerative diseases, vascular disorders (hemorrhaging and blood clots) and possibly reproductive issues. There is also the risk that the vaccines will accelerate the emergence of new strains of the virus that are no longer sensitive to the antibodies produced by the vaccines. When people are immune compromised (e.g., taking chemotherapy for cancer), the antibodies they produce may not be able to keep the virus in check because the immune system is too impaired. Just as in the case of antibiotic resistance, new strains evolve within an infected immune-compromised person's body that produce a version of the spike protein that no longer binds with the acquired antibodies. These new strains quickly come to dominate over the original strain, especially when the general population is heavily vaccinated with a vaccine that is specific to the original strain. This problem is likely going to necessitate the repeated rollout of new versions of the vaccine at periodic intervals that people will have to receive to induce yet another round of antibody production in an endless game of cat and mouse.

Like the mRNA vaccines, the DNA vaccines are based on novel biotech gene editing techniques that are brand new, so they too are a massive experiment unleashed on a huge unsuspecting population, with unknown consequences. Both DNA vector vaccines have been associated with a very rare condition called thrombocytopenia, in which platelet counts drop precipitously, resulting in system-wide blood clots and a high risk of cerebral hemorrhaging [5]. This is likely due to an autoimmune reaction to the platelets, and it comes with a high risk of mortality. In the case of the AZ vaccine, this has caused over 20 European countries to temporarily pause their vaccination programs [6]. And the United States called a temporary halt on the J&J vaccine.

fascinating theory to explain this depends on the fact that DNA vector vaccines require the DNA to be copied into RNA in the nucleus, and this presents the possibility of producing an incomplete copy, generated through "splice variants," that is missing the code for attaching to the membrane. These soluble partial sequences wander off to other parts of the body and bind to ACE2 receptors throughout the vasculature. Antibodies to these ACE2-bound partial spike fragments cause an acute inflammatory response that results in disseminated intravascular coagulation (DIC).

How to Make an Adenovirus DNA Vector Vaccine

The adenovirus vaccines are created through techniques that the average citizen can't possibly fathom could even exist. For the AZ vaccine, the bulk of the DNA in the vaccine codes for the various proteins that are needed by a strain of adenovirus that mainly infects chimpanzees and causes cold-like symptoms. However, it is not a "normal" version of this cold virus. First of all, it has been stripped of certain genes that it needs in order to replicate, and for this reason it is referred to as an "adenovirus vector." This defect, it is argued, keeps it from actually infecting the vaccinated patient. Secondly, it is modified, through gene editing techniques, to create a recombinant version of the virus that contains the complete coding sequence for the SARS-CoV-2 spike protein, spliced into its DNA sequence – the same protein that the RNA vaccines code for. The recombinant DNA is a linear double-stranded DNA sequence where proteins from two different species are integrated through gene editing.

But they solved this problem by making use of a genetically modified version of a human cell line, called HEK (human embryonic kidney) 293 cells, where the human cell's DNA was transfected long ago with fragments of the genome of an adenovirus – conveniently providing the defective recombinant virus with the missing proteins it needs to be able to proliferate. Within a culture of these HEK 293 cells, the virus can replicate, assisted by the proteins that are produced by the host cells. The HEK 293 cells originally came from a kidney of an aborted fetus, and it has been maintained in culture ever since the 1970s, because it was modified to become immortal, with the help of the adenovirus. Although it was obtained from a kidney, it is not a kidney cell. In fact, it has many properties that are characteristic of a neuronal stem cell. The fact is, they don't really know what kind of cell it is. The ability of a cell line to survive indefinitely is a feature of tumor cells. Although the vaccine is "purified" during the processing, there is no guarantee that it is not contaminated with remnants from the host cells, i.e., human DNA of a neuronal tumor cell line. It does not seem like a good idea to inject the DNA of a human tumor cell into anyone.

The J&J vaccine has a very similar manufacturing process, except with a different adenovirus strain and a different human host cell. For J&J, the host cell is another fetal cell line harvested long ago and made immortal through the incorporation of adenovirus genes into the host human genome. This cell line was taken from the retina of the eye of the fetus.

The Spike Protein is Toxic

protein that is the main constituent of the SARS-CoV-2 protein cage that encloses its RNA contents. Both the DNA vector and the RNA vaccines induce the vaccine-infected cell to manufacture many copies of the spike protein according to the code. Through experimentation, researchers have determined that the spike protein is toxic even when introduced all by itself. In a revealing experiment, researchers injected spike protein into hamsters, and found that it was taken up by endothelial cells lining the blood vessels, via ACE2 receptors. This caused a downregulation of ACE2, which had significant effects on the metabolic policy in the cells. In particular, it inhibited the synthesis of mitochondria, and caused the existing mitochondria to fragment. Mitochondria are the organelles in the cell that produce large quantities of ATP (the energy currency of cells) by oxidizing nutrients, while consuming oxygen and producing water and carbon dioxide. The spike protein reduced the production of ATP by mitochondria and increased glycolysis – the alternative, much less efficient, way to produce ATP without using oxygen. This metabolic change towards getting energy through glycolysis is a characteristic feature of cancer cells and of neurons in neurodegenerative diseases such as Alzheimer's.

In another experiment, researchers showed that spike protein can cross the blood-brain barrier in mice and be taken up by neurons throughout the brain. This too is likely mediated by ACE2 receptors (which neurons also produce). These same researchers also showed that spike protein administered in the nose was able to reach the brain by traveling along the olfactory nerve. When they induced inflammation in the brain through exposure to lipopolysaccharide (LPS), they saw an increased uptake of spike protein into the brain, which they hypothesized was caused by increased leakiness in the barrier. As you will see, these points become important when we later consider what happens following a SARS-CoV-2 vaccine, which is designed to induce inflammation.

Many people suffering from COVID-19 have experienced symptoms characteristic of the central nervous system such as headache, nausea, dizziness, fatal brain blood clots and encephalitis. In an advanced 3D microfluid model of the human BBB, researchers in the United States showed that the spike protein by itself disrupts the blood brain barrier by inducing an inflammatory state, and they proposed that this could be the source of such symptoms.

brain. ACE2 was expressed in astrocytes, pericytes (cells that wrap around the endothelial cells lining capillary walls) and in endothelial cells – and all of these are key components of the blood-brain barrier. Perhaps of even greater concern is that ACE2 was highly expressed in the substantia nigra, a brain-stem nucleus where damaged dopaminergic neurons lead to Parkinson's disease.

Bell's Palsy, Autism and Parkinson's Disease

In a paper aptly titled, "Is COVID-19 a Perfect Storm for Parkinson's Disease?" researchers made a strong case for the possibility that we will see an increase in Parkinson's disease in the future, due to the COVID-19 pandemic. They refer to three separate cases where acute Parkinsonism developed shortly after a COVID-19 infection. They proposed that systemic inflammation caused by severe COVID-19 could trigger neuroinflammation in the substantia nigra, killing off dopaminergic neurons. These neurons express high levels of the ACE2 receptor, making them highly vulnerable to the spike protein. A viral infection is known to upregulate α-synuclein, which, in high concentrations, forms soluble oligomers that then precipitate out as fibrils and accumulate within "Lewy bodies" that are tightly linked to Parkinson's disease. Further corroboration of this idea comes from a paper which demonstrated that an infection with SARS-CoV-2 causes brain inflammation in macaques and induces the formation of Lewy bodies.

Parkinson's disease is the second most common neurodegenerative disorder and the most common neurodegenerative motor disorder. The root cause of nearly 90% of cases remains unknown, but it has been theorized that viral infections are often involved. It can be argued that the loss of a sense of smell and/or taste in association with COVID-19 is a sign of a Parkinsonian link, since this symptom is also an early sign of Parkinson's disease.

"waking up" and causing disease symptoms. This observation is based on the fact that shingles and facial palsy (Bell's palsy) are being commonly reported in side-effect reports in the FDA's Vaccine Adverse Event Reporting System. As of May 21, 2021, over 2500 reports of Bell's palsy following COVID-19 vaccines had appeared in VAERS. A primary cause of Bell's palsy is the activation of latent viral infections, most notably Herpes simplex and Varicella zoster, Varicella zoster is also the virus responsible for shingles.

While Bell's palsy usually resolves over time, there can be some serious longer-term consequences. Pregnant women who are diagnosed with active herpes infections during pregnancy have a 2-fold increased risk of having an autistic male child from that pregnancy. This should make a pregnant woman hesitate to get a SARS-CoV-2 vaccine. Bell's palsy can also be a risk factor for Parkinson's disease much later in life. A study on nearly 200 Parkinson's disease patients compared with age- and gender-matched controls found that six of the Parkinson's patients had had an earlier diagnosis of Bell's palsy, whereas none of the control patients had. There's also a link between autism and Parkinson's disease. A study on autistic adults over 39 years old found that one third of them had symptoms that meet the criteria for a Parkinson's diagnosis.

Prion Diseases

Prion diseases are a group of severe neurodegenerative diseases that are caused by misfolded prion proteins. The most common prion disease in humans is the always-fatal sporadic Creutzfeldt-Jakob disease (CJD), which accounts for more than 85% of the cases. Prion diseases are more specifically called transmissible spongiform encephalopathies (TSEs), and infection can spread through exposure to misfolded proteins as "infective" agents, without requiring a live pathogen. PrP is the name given to the specific prion protein associated with these TSEs. Misfolded PrP proteins act as a seed or catalyst that then recruits other molecules of PrP to misfold in the same way and glom together into pathogenic fibrils.

the 1990s, is probably the best-known TSE. While eating beef from an infected animal is a very rare risk factor, most cases of Creutzfeldt-Jakob disease occur for unknown reasons, and no other risk factors have been identified. A study based in Switzerland confirmed that many patients who died of Creutzfeldt-Jakob disease had detectable levels of a prion protein in their spleen and muscles, in addition to the olfactory lobe and the central nervous system. More generally, diseases involving misfolded PrPs have consistently been found to involve an initial early phase of prion replication in the spleen which happens long before overt symptoms appear. This point becomes important when we consider whether the COVID-19 vaccines might cause prion diseases.

PrP has a unique feature that it contains multiple copies of a characteristic motif in its amino acid sequence that is called a "GxxxG" motif, also known as a "glycine zipper". These proteins normally fold into a characteristic shape called an alpha helix, which allows the protein to penetrate the plasma membrane. The glycines in the zipper motif play an essential role in cross-linking and stabilizing alpha helices. This glycine zipper motif is also a common characteristic of many transmembrane proteins (proteins that cross the membrane of the cell).

Indeed, the coronavirus spike protein has a GxxxG motif in its transmembrane domain (specifically, GFIAG – glycine, phenylalanine, isoproline, alanine, glycine). There is a platform called "Uniprot" where you can look up the sequence of specific proteins. The Uniprot entry for the SARS-CoV-2 spike protein has five glycine zipper sequences altogether. According to J. Bart Classen, the SARS-CoV-2 spike protein has the ability "to form amyloid and toxic aggregates that can act as seeds to aggregate many of the misfolded brain proteins and can ultimately lead to neurodegeneration."

prion-like properties, and these diseases are characterized as protein-misfolding diseases or proteopathies. Like PrP, prion-like proteins become pathogenic when their alpha helices misfold as beta sheets, and the protein is then impaired in its ability to enter the membrane. These diseases include Alzheimer's, amyotrophic lateral sclerosis (ALS), Huntington's disease and Parkinson's disease, and each of these is associated with a particular protein that misfolds and accumulates in inclusion bodies in association with the disease. We already saw that Parkinson's disease is characterized by Lewy bodies in the substantia nigra that accumulate misfolded α-synuclein.

Glycines within the glycine zipper transmembrane motifs in the amyloid beta precursor protein (APP) play a central role in the misfolding of amyloid beta linked to Alzheimer's disease (Decock et al., 2016). APP contains a total of four GxxxG motifs (one fewer than the spike protein).

A case study presented the case of a man who developed CKD simultaneously with symptomatic COVID-19. The authors proposed that infection with SARS-CoV-2 precipitates or accelerates neurodegenerative diseases. A theoretical paper published by researchers in India showed that the spike protein binds to a number of aggregation-prone prion-like proteins, including amyloid beta, α-synuclein, tau, PrP and TDP-43. They argued that this could initiate aggregation of these proteins in the brain, leading to neurodegeneration.

Tracing the Vaccine Trail to the Spleen

It is important to understand what happens to the contents of a vaccine after it is injected into the arm. Where does it travel in the body, and what does it do in the places where it settles in?

Vaccine developers are keen to know whether the vaccine induces a strong immune response, reflected in high antibody production against the spike protein, in the case of COVID-19 vaccines. And to do this, they need to trace its movement in the body.

virus. They detect an immune complex with viral proteins that are exposed on the surface of an infected cell. A study on an adenovirus-vector based vaccination of mice used clever methods to produce a marker that could track the activity of CD8+ T-cells in the lymph system and the spleen, in the days following vaccination. It can be inferred that immune cells (antigen-presenting cells, where the "antigen" is the spike protein) were initially present at the arm muscle injection site and synthesized the virus spike protein from the vaccine DNA code, exposing it on their surface. Once activated by the foreign protein, they translocated into the draining lymph nodes and finally made their way to the spleen via the lymph system. The CD8+ T-cells are idly waiting within the lymphatics until they spot an infected immune cell. Researchers could detect activation of CD8+ immune cells over time and inferred that this was caused by the arrival of the contents of the vaccine to the site where these immune cells reside. Activated CD8+ T-cells first appeared in the draining lymph nodes, but after five days began to show up in the spleen. Their numbers there peaked sharply by 12 days and then remained high with a slow decay up to 47 days, when the researchers stopped looking. What this means is that the vaccine is picked up by antigen-presenting cells at the injection site and carried to the spleen via the lymph system. The carrier cells then hang out in the spleen for a long time. And this is where the danger lies in terms of the potential to cause prion disease.

In the paper that Greg Nigh and I published recently on the mRNA vaccines, we argued that the mRNA vaccines are rather perfectly set up to produce a very dangerous situation in the spleen that is poised to launch a prion disease. Given the fact that the DNA vector vaccines also end up concentrated in the spleen, I think that the same thing holds true for them as well. The spleen is where the action is for seeding misfolded prion proteins. The vaccine-infected cells have been programmed to produce large amounts of spike proteins. Prion proteins misfold into damaging beta-sheet oligomers when there are too many of them in the cytoplasm. Might the spike protein do the same?

Europe (Pfizer, Moderna, and J&J) use a genetic code for the spike protein that has been slightly tweaked, in order to produce a more potent antibody response. Normally, after binding to the ACE2 receptor, the spike protein spontaneously changes its shape in a dramatic way in order to fuse with the membrane of the cell. In a Web publication, Ryan Cross described this action very graphically based on a spring-like model, as follows: "When the spike protein binds to a human cell, that spring is released, and the two helices and the loop straighten into one long helix that harpoons the human cell and pulls the virus and human membranes close together until they fuse." As Cross explains, through trial and error, but taking structural information into account, researchers came up with the idea of swapping out two adjacent amino acids for prolines in the membrane fusion domain in order to stabilize the shape of the spike protein in its pre-fusion form. In this form, it exposes critical antigenic areas, and this assures more rapid formation of matching antibodies, the only goal of the vaccine design. This also prevents the protein from fusing with the plasma membrane of a host cell. I'd imagine that the spike protein attaches to the ACE2 receptor and then gets stuck there, like a sitting duck. But a worrisome thought is whether this open state, not fused with the membrane, might more closely resemble the shape of a misfolded prion-like protein like amyloid beta than does the collapsed shape it needs to go into the membrane?

Tetz and Tetz have argued in a published online preprint that prion-like domains in the spike protein enable higher affinity for the ACE2 receptor, making the virus more virulent than its earlier cousins. These same authors published an earlier peer-reviewed journal paper where they observed that many other viruses have proteins in their coat that have distinct features of prion proteins.

Germinal Centers and Parkinson's Disease

Germinal centers in the spleen are a primary factory where antibodies against specific antigens (such as the spike protein) are manufactured and perfected. Makers of the mRNA vaccines were pleased to see that antigen-presenting cells (mainly dendritic cells), originally attracted to the site of the injection, take up the mRNA particles and then migrate via the lymph system to the spleen in high numbers and induce high levels of antibody production in these germinal centers.

process of producing and distributing misfolded prion proteins, often seeded by viral proteins, and triggered by an acute inflammatory response.

B cells, also known as B lymphocytes, are a type of immune cell that is the key player in the process that leads to the production of specific antibodies to a foreign antigen [38]. They originate from precursor cells in the bone marrow, and then migrate to the spleen and other lymphoid organs, where they bind to antigens presented to them by antigen-presenting cells, such as the dendritic cells. A maturation process beginning with a multipotent progenitor B cell ends with a mature "memory" B cell that has gone through a complex process to perfect its antibody production process to specifically match the antigen it has been assigned to (e.g., the spike protein). B cells also go through another process called class switching, which changes the type of antibody they produce from one class to another, without changing its specificity to the antigen.

Antibodies are also known as immunoglobulins (Igs), and the possible classes include IgM, IgG, IgA and IgE. IgM is the first immunoglobulin class that is produced (primarily in the spleen), and it is converted into IgG through class switching. IgG is the dominant class in the blood, making up 75% of the serum antibodies, and it is essential for clearing infections in the tissues. Long-lived mature memory B cells cruise the blood stream looking for any appearances of the antigen they have been assigned to, but they are useless for anything else. When the virus they've been trained to match mutates to the point where their antibodies no longer match well, they become useless even for the disease they're trained to fight.

When mice are injected with PrP in the abdomen (intraperitoneal injection), the PrP shows up very quickly in the spleen. From there, the PrP travels along the spinal cord and the vagus nerve to reach the brain, causing prion disease [39]. As we will soon see, α-synuclein, the prion-like protein linked to Parkinson's disease, also makes its way to the brain from the spleen along the vagus nerve. The mRNA vaccines set up perfect conditions in the spleen for the formation and distribution of conglomerates made up of misfolded α-synuclein, PrP and spike protein.

shape it is an active participant in the immune response. α-Synuclein facilitates the processes that lead to antibody production in response to foreign antigens. Dendritic cells express α-synuclein, and it is upregulated (over-expressed) in response to stressors, such as the mRNA, the cationic lipids, and the PEG in the mRNA vaccines. Much can be learned by studying mice that have been genetically engineered to have a defective version of α-synuclein. These mice have a decreased capacity to clear pathogens through phagocytosis, and an impairment in the ability to generate B cells from precursor stem cells. They also had a four-fold reduction in progenitor B cells in the bone marrow. The amount of immunoglobulin G was reduced compared to wildtype, suggesting impaired class switching. Altogether, they are unable to mount an effective immune response to antigens, whether they come from a natural threat or a vaccine.

Dendritic cells under stress accumulate prion proteins and release them into small lipid particles called exosomes, which are then distributed throughout the body, either along nerve fibers or in the general circulation. There is reason to believe that these vaccines will accelerate the release of exosomes containing misfolded prion-like spike proteins that are being produced in large amounts under instruction from the vaccines. These spike proteins will act as seeds to cause α-synuclein and PrP to also misfold and form toxic oligomers together with the spike protein, which are released into the extracellular space as exosomes. These exosomes, released under the severe stress conditions induced by the vaccine, then carry prion proteins into the brain along the vagus nerve, to initiate prion diseases.

Impaired Immune Response due to Over-vaccination

new pathogenic threats, and this is reflected in a failure to generate protective antibodies in response to vaccination. It has been demonstrated in experiments with mice that aged mice have an overabundance of long-lived memory (antigen-experienced) B cells, and this is paired with an inability to generate new B cells from progenitor cells in the bone marrow, as well as impairment in the process of refinement of the antibody response in germinal centers in the spleen and the associated class switching that produces effective IgG antibodies. A significant reduction in the number of naive follicular B cells, combined with an impaired ability to convert them into mature memory B cells leaves these aged mice highly vulnerable to new infections. It is likely that the same principle applies to humans. A plausible conclusion is that aggressive vaccination campaigns accelerate the pace at which an individual's immune system reaches an "aged" status due to exuberant generation of memory B cells in response to the artificial stimuli induced by repeated vaccination.

It has now been confirmed that the S1 component of the spike protein shows up in the blood one day after the first mRNA vaccine and remains detectable for up to a month after vaccination, becoming cleared as IgA and IgG antibodies become available. For immune compromised people, it likely stays in the blood much longer, exposing all the tissues – the spleen, the heart, the brain, the gonads, etc. – to the toxic prion-like spike protein.

Today's children are by far the most vaccinated generation in the history of humankind. If we decide in the near future to deliver a booster COVID-19 shot to them every year, as seems possible given the current climate of enthusiasm for these vaccines, are we inviting disaster for them in years to come? Will their immune system "age" much faster than that of previous generations, due to the exhaustion of the pool of progenitor B cells by all these vaccines? Will they succumb to Parkinson's disease or other debilitating prion-based neurodegenerative diseases much sooner and in much greater numbers than previous generations? This is an experiment that I hope we finally decide not to carry out.

Summary

rushed to market with grossly inadequate evaluation and aggressively promoted to an uninformed public, with the potential for huge, irreversible, negative consequences. One potential consequence is to exhaust the finite supply of progenitor B cells in the bone marrow early in life, causing an inability to mount new antibodies to infectious agents. An even more worrisome possibility is that these vaccines, both the mRNA vaccines and the DNA vector vaccines, may be a pathway to crippling disease sometime in the future. Through the prion-like action of the spike protein, we will likely see an alarming increase in several major neurodegenerative diseases, including Parkinson's disease, CKD, ALS and Alzheimer's, and these diseases will show up with increasing prevalence among younger and younger populations, in years to come. Unfortunately, we won't know whether the vaccines caused this increase, because there will usually be a long time separation between the vaccination event and the disease diagnosis. Very convenient for the vaccine manufacturers, who stand to make huge profits off of our misfortunes – both from the sale of the vaccines themselves and from the large medical cost of treating all these debilitating diseases.

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