Introduction

As most of you probably know, the 2023 Nobel Prize in medicine was awarded to two researchers whose patents were licensed by Pfizer & Moderna in the creation of the mRNA injectable products.

This is significant since the Nobel Prize still holds a good bit of sway. Questionable and hotly debated factual claims can step-by-step become historical truth through these sorts of public rituals.

At the same time, this last year alone has revealed quite a few details about the risks connected to these pharmaceutical products and the specific causal pathways through which their detrimental effects obtain or potentially obtain. In other words, we now know much more about what they actually do, about what likely lies behind the obviously damning statistics and tragedies that these last few years have so amply provided.

Brief overview of the problems of the covid mRNA vaccines

Many other writers and researchers have spelled out everything in much greater detail than I'm able, so I'll just try to provide a clear and relatively concise overview that gives you the most important facts of the matter.

In summary, the covid mRNA vaccines can plausibly be connected, via several evident, separate, and likely synergistic causal mechanisms (rather than just through the epidemiological correlations), to heart disease, clotting and thrombotic events, cancer, immunity & autoimmunity issues, neurological issues, reproductive problems, an immunological priming that creates susceptibility to future infections, and finally, they also engender a non-sterilizing immunity that promotes rapid viral evolution with problematic tendencies among the general population.

One important to note before we begin is that the effects of these products are going to be somewhat erratic.

We're dealing with a complex cocktail of biologically active, synthetic substances that interact with the human body on several levels, and likely also in synergistic ways (i.e. the ingredients, when combined, bring about additional effects beyond that of the separate substances).

This can be contrasted to something like insulin, which is a single substance familiar to the body that interacts with us in a predictable way when it is injected.

Complex, synergistic effects and medical data

Why is this relevant? With erratic and complex negative effects, it's harder to establish clear and unambiguous correlations through empirical data since the signals get fuzzier. For example, if a treatment increases cancer risk in the short term by 250% in every patient, any basic sampling of the patient data will strongly indicate this.

But if a treatment rather inflicts one or more negative health effects from a whole plethora of alternatives over a longer timeline, it's not going to be quite as obvious. Even if every single patient is strongly negatively affected over time, the full extent of the damage will be harder to discern since even something like an RCT will have to select a few specific variables.

So let's say you look for a cancer signal in the patient data, and you find a weak one. If you settle for that, case closed, and there still remains a vast variety of pathways for damage to uncover, maybe you've just missed 99 out of 100 negative health outcomes.

Ironically, a treatment that cumulatively does many times more damage than a less harmful substance will be harder to recognize as unsafe if the negative effects 1) are individually moderate, 2) come through a wide variety of pathways, and 3) are slower to manifest. The less damaging substance might be over the threshold for a minor increase in cancer risk throught the population, while the more harmful treatment is just below the threshold on hundreds of variables, even though its effects get progressively worse and the treatment on the whole is vastly more dangerous.

You should be able to see the signs of damage in the broad-scope epidemiological data, but the specific connections will take more work to pin down in detail, and they will be easier to ignore since there are so many potential confounding variables (you know, kids get strokes too, and climate change fear causes heart disease).

Compounding this are also the facts that there's variation between the vaccine batches (different levels of degradation of the mRNA, for instance); different distribution methods (some were given the vaccine intravenously); as well as different immunity environments for everyone injected.

In short, there are a lot of parameters involved, but now, there's an accumulated amount of detailed data to paint a rather clear picture of the etiological mechanisms behind the negative effects of the mRNA vaccines.

The contents of the vaccines, their effects and their biodistribution

This complexity of the vaccines' effects were brought to my awareness back in 2021, when our research networks tried to make sense of the emerging data indicating detrimental effects, such as the VAERS signals. It was immediately observed that the vaccines involve at least **three major kinds** of foreign substances introduced into the body, each with complex effects on the human being - the synthetic nanolipids, the "preservatives" stabilizing the mRNA, as well as the specific mRNA payload itself and the spike protein it generates.

The vaccine does not stay at the site of injection

From the data on the biodistribution of the LNPs and their payload, we also know that this set of foreign substances enter the bloodstream and also accumulate in the liver, spleen, adrenals and the ovaries - following the straightforward and recommended intramuscular (and not intravenous injection).

There's even recently published unambiguous evidence that vaccine mRNA is generally detectible even in the breast milk of lactating women within hours of the inoculations (Hanna et al. 2023) which should finally put an end to the discussion on whether or not the vaccine normally remains at the injection site. To get into the breast milk, the LNPs will have to travel through the vascular or lymphatic system, which of course is to be expected from the Japanese FOIA data, the recently released report from the Australian Dept. of Health (2021), and the earlier biodistribution experiments.

So what does this mean? Well, the LNPs and their payload do not remain at the site of injection. Marc Girardot argues that they "trickle back" into the bloodstream via the lymphatic system even if they're administered into muscle tissue, which is plausible. They are nonetheless evidently distributed throughout the tissues in the body via the bloodstream or the lymphatic vessels, and they accumulate in certain important organs. You mainly find them in the blood and the filtering organs.

This cannot be over-emphasized. The vaccine LNPs were not supposed to get into the bloodstream, and there's now unambiguous, mainstream evidence that they generally do.

Which are then these component substances of the vaccine in detail, and what are their likely or inevitable effects on the tissues they interact with and wherein they bioaccumulate?

Synthetic nanolipids

The synthetic nanolipids (or lipid nanoparticles, LNPs) are basically small globs of partially synthetic fat that encapsulate the mRNA so it can be distributed to the ribosomes and produce the spike protein for immunization.

In the covid vaccines, the LNPs consist of four separate types of fats or fat-like substances: cationic lipids, polyethylene glycol, phospholipids and cholesterol (Wilson & Geetha 2022).

The main problems of the LNPs or synthetic nanolipids are toxicity issues (mainly the cationic lipids), allergy issues (anaphylaxis from the polyethylene glycol), immune system dysregulation, and the fact that the LNPs and their payload tend to accumulate in the liver, spleen, adrenals and in the reproductive organs.

This is a quite complex chapter in and of itself which not least Jessica Rose has dug deeply into. Her work is highly recommended for details and further references.

To begin with, two of the compounds in the LNPs have a significant toxicity profile by themselves, and there's a long list of potential chemical interactions that could promote further toxic effects in vivo, i.e. in actual human subjects.

This LNP delivery system is both quite new, and involves a lot of moving parts. The for-profit pharmaceutical industry first began to investigate them as a vehicle for drugs back in 2005, so there has not been much time to evaluate their effects. Due to the complexity of the compounds, proper evaluations would also have been both costly and technically difficult.

Let's just say that there's a low chance for this being a top priority for venture capital in the pharmaceutical field.

Toxicity and the effects of transfection

Soenen, Brisson & De Cuyper (2009) detail specifically how the versatility of the synthetic cationic lipids had rendered them being important components in all sorts of biomedical research, but that this functionality (and potential profitability) also led to toxicity issues being ignored.

There were indeed clear signs of danger in the early development phases more than 17 years ago (see e.g. Lv et al. 2006), and a 2018 article emphasizes the cytotoxic (toxic for cells) effects of cationic lipids used for gene delivery as much more significant than those of gasoline (Cui et al. 2018, cf. Sayyed et al. 2022).

As a side note, Mahmoodpoor et al. (2012) provide a fun case study about what happens when you inject 10 mls of gasoline into the vascular system, but the amounts of cytotoxic substance in the covid shots are almost incomparably miniscule in contrast, so it's not for comparison.

Cardiovascular and thrombogenic effects

The cytotoxic effects of the LNPs are still an important causal factor in the etiologies we see here, however. They are particularly important in connection to the cardiovascular system and the etiology of heart disease, clotting and thrombotic events. The endothelium (the smooth lining of the inside of the blood vessels) is sensitive not only to cytotoxicity, but also to the vaccines' intended mechanism of transfection (the process whereby they introduce the foreign mRNA into the ribosome to generate the S-protein). Again, this risk was supposed to be minimal since the LNPs were said to remain at the site of infection.

With regard to cytotoxicity, it's well known that chemotherapy in cancer treatment, where the cytotoxic effects are fully intentional, cause heart disease (e.g. myocarditis), thrombotic events, congestive heart failure and such. This is not least due to the cytotoxic effects on the lining of the blood vessels, which trigger inflammation and the body's cellular repair processes (Shakir 2009). These are the well-known causal pathways to such conditions as atherosclerosis, congestive heart failure, and various heart-related inflammatory events.

The other separate mechanism by which the LNPs cause problems with the endothelium (the blood vessel lining) is the transfection of the cell - the introduction of the mRNA for production of the spike protein. If the LNPs are non-specific in terms of what cell types they target for transfection (which is strongly implied by Hanna et al. and what they found regarding the introduction of mRNA into breast milk), then the endothelial cells throughout the body will by far be the most common target for transfection since the overwhelming majority of contact points between the LNPs and the body's cells will take place in the vascular system. Simply put, the lipid nanoparticles are distributed throughout the body, and in this process, it's mostly the endothelial cells that they interact with.

It's also worth to note that the vascular system distributes material throughout the body incredibly rapidly. If you inject something intravenuously, it's all over the body in a matter of seconds, and whatever you inject will quickly interact with the endothelium at billions of contact points, by billions of LNPs.

Anyway, transfection by the mRNA payload also causes inflammation.

Transfection is the intended process by which the mRNA is introduced into the cell to generate the immunizing spike protein, but it's also going to damage and eventually kill the host cell, specifically by necrosis or apoptosis. If this briefly happens in the muscle tissue at the site of inoculation, it's no big deal even if the inflammation persists for some time. If it takes place in the endothelium it can trigger cardiovascular disease.

Another important point of note is that transfection and problems related to cytotoxicity will be increasingly likely in smaller vessels, such as capillaries. Here's not least where Marc Girardots "bolus theory" comes in, which purports to explain most adverse events by the unintended injection of the mRNA products directly into the bloodstream. In terms of CV events, the mechanism would basically be that you get a sort of viscous lump of injection fluid that massively increases the local transfection rate and cytotoxic effects in tighter vessels, thus potentially generating significant localized inflammation. Strong localized inflammation is far worse, since the body can handle quite a lot of evenly distributed inflamed cells throughout the endothelium without anything like clotting problems or massive necrosis occuring.

It's also worth noticing that anti-spike antibodies also have cytotoxic effects on top of all this, likely compounding the cytotoxic effects of the LNPs and the transfection processes as such (Phan et al. 2023).

Other negative health outcomes

The above also provides a mechanism of action for organ damage, myocarditis, necrosis, clotting and thrombogenic events, and in terms of penetration of the various blood barriers, many types of neurological problems, fertility issues and immunity suppression.

The specific accumulation of the LNPs in the female reproductive organs could also plausibly impact female fertility through cytotoxicity. It's well known that chemotherapy impacts female fertility, both by triggering ovarian failure and degrading the health of the oocytes (Sonigo et al. 2019).

While the covid mRNA vaccines don't immediately impact ovarian reserve (Soysal & Yilmaz 2022), it's hard to see how bioaccumulation and persistence of the cytotoxic LNPs and anti-spike antibodies in the reproductive organs would have no consequences for the viability of the oocytes, especially in light of the penetration of the blood-tissue barriers discussed in more detail in relation to other modalities of harm later.

Pseudouridine (N1-Methylpseudouridine) and codon optimization

The synthetic pseudouridine is used as part of the LNP as a sort of preservative. Its intended function is to stabilize the mRNA so that it doesn't decay, and can be effectively transmitted into the ribosomes, which then produce the immunizing spike protein.

The main issue with the N1-Methylpseudouridine is that it disrupts the mRNA translation process through something called frameshifting, causing the ribosomes to produce other things besides spike, while also destabilizing the proteins manufactured by the ribosomes. Think of it as glitches in the code that causes a randomly faulty output (Wiseman et al. 2023).

This is further compounded by the fact that the specific modifications of the mRNA molecule through what's known as "codon optimization" which serves to fine-tune the mRNA translation (so that maximum amounts of spike are produced in the ribosomes) also increase translation errors. This might sound contradictory, but it's simply the case that while maximizing spike output, the production of faulty, unstable and defective proteins can also be increased (Mauro & Chappell 2014).

So what are the potential consequences of this tandem impact on the mRNA translation process? Well, the massive introduction of billions of LNPs with their codon-optimized payload and synthetic pseudouridine can trigger aberrant protein production and the misfolding of proteins (Xia 2021) in the transfected cells.

Instead of spike, a significant number of transfected cells will produce garbage proteins, junk data. This is a potentially huge problem for several reasons.

As anyone who was around back in the 90s will recall, prion-related diseases like Creutzfeldt-Jacob disease ("mad cow disease"), but also Alzheimer's, Parkinson's, certain forms of diabetes, MS, and many other conditions, are manifest through the cascading misfolding of prion proteins. Cascading means, in other words, that the mere presence of misfolded prions will cause further misfolding in other proteins, reproducing the problem (Wickner et al. 2023).

This means that you don't need an indefinite presence of ribosomal spike protein factories, as the misfolded proteins can and will remain active after the initially transfected cells have died off.

Misfolded prions also accumulate naturally, which means that even a uniformly distributed presence of billions of LNPs throughout the body can potentially trigger pathogenesis (Lambert et al. 2021).

And on top of that, a peptide of the coronavirus spike protein (a less complex amino acid formation closely related to the spike protein), has been shown to exhibit amyloidogenic properties. So relatively minute changes in the structure of the spike protein that plausibly can be generated by translation errors and/or degraded mRNA will generate a peptide that has the propensity to create amyloid structures ("amyloid nanotape structures") in the body (Castelletto & Hamley 2022).

What's extra noteworthy is that this peptide, like the spike protein, will target the ACE-2 receptors, so it will preferentially bind to heart, lung and endothelial tissue (Guney & Akar 2021; Shirbhate et al. 2021).

The mRNA translation problems thus provide a causal mechanism for various pathological conditions in addition to the basic inflammatory and cytotoxic issues we can connect to the LNP as such. It also explains the emergence of disease apart from and in addition to the mechanisms described by the bolus theory, i.e. the LNPs can through translation problems trigger various sorts of illness even without any significant local inflammation and cytotoxicity.

There's also a pathway towards exacerbating autoimmune conditions from this junk protein production. We already know that the spike protein has a significant potential to generate autoimmune responses since there are marked similarities between human proteins and spike (Nunez-Castilla et al. 2022). The authors of this paper emphasize that spike and many human proteins share antibody-binding properties, which implies a potential cross-reactivity between human and spike protein by the anti-spike antibodies - autoimmune reactions.

If you then massively generate slightly off-center spike proteins and spike-like peptides, the risk is that many of these proteins will accentuate this cross-reactivity with human proteins, causing autoimmune fixations of the immune system.

There's also a connection between the spike protein and fertility-related proteins which looks especially alarming in the context of autoimmunity:

> *SARS-CoV-2 spike glycoprotein was found to share 41 minimal immune determinants, that is, pentapeptides, with 27 human proteins that relate to oogenesis, uterine receptivity, decidualization, and placentation. All the shared pentapeptides that we identified, with the exception of four, are also present in*

> *SARS-CoV-2 spike glycoprotein-derived epitopes that have been experimentally validated as immunoreactive (Dotan et al. 2021).*

Lake & Breen (2023) also make similar observations in relation to MS-related proteins.

It's worth noting that these autoimmune red flags would not have been much of an issue without the spike protein entering the bloodstream in massive amounts, i.e. in the context of an upper respiratory tract infection by the covid virus, these sorts of risks would be negligible.

But you don't want huge amounts of this in the vascular system, getting distributed throughout the body's tissues.

The mRNA payload and the spike protein

We covered certain aspects of the mRNA in the section above, but I'll add a number of further observations specific to the mRNA further down.

So we know that the spike protein is not going to be strongly expressed in the vascular system through normal infection of the lungs and airways. This was observed back in 2020 by Trypsteen et al., who found that you, just as one would expect, get the virus colonizing the upper airways, the mouth and the lungs, and then find it in the GI tract and the urinary system through excretion - with only about 6% of the virus actually detectible in the blood, and likely not in a very viable form. SARS-CoV-2 specifically replicates in the upper respiratory epithelia where they can bind with the ACE2 receptors (V'kovski et al. 2020) and does not readily replicate in the bloodsteam. Basically, covid is not a blood infection.

It's even a bit iffy whether it actually can effectively replicate in the blood at all, but in any case, the vascular system is obviously not normally going to be a main reservoir for the virus and is not a favoured enviroment for replication:

> *A recent study demonstrated that SARS-CoV-2 can infect CD4+T cells but does not actively replicate within the host T cells. Case reports showed that haematopoietic cell transplantation (HCT) from pre-symptomatic donors with a viral RNA-positive nasopharyngeal sample* ***did not cause COVID-19 in the recipient****. The absence of SARS-CoV-2 transmission via HCT or blood transfusion, and the uncertainty surrounding the possibility of virus replication in HSC, suggest that the risk of COVID-19 transmission by HCT and chimeric antigen receptor T cells (CAR T-cells) therapy is [merely] theoretical (Hegarty 2020).*

This contrasts significantly with the situation after inoculation with the mRNA vaccines. As we saw above, the LNPs mainly get into the blood and the filtering organs.

The mRNA is detectible in the blood up to at least 15 days after vaccination where it travels freely, protected by its handy LNP shield (Fertig et al. 2022), and is able to interact with the endothelial cells in the vascular system, transfecting them to generate the spike protein.

Spike pathologies

Recent evidence underscores the spike proteins' role in causing vascular disease (Perico, Benigni, Remuzzi 2023), and thus their role in the etiologies of thrombotic events, strokes, hypertension, myo- and pericarditis &c.

Yonker et al. (2023) also show how there's persistent free spike protein in the blood of post-vaccination myocarditis patients and NO free spike in the vaccinated but asymptomatic controls. As an aside, this seems consistent with Girardots bolus theory, i.e. that significant localized transfection (and inflammation) generates continuous production of spike protein whose concentrations also increase the likelihood of damage:

> *… a growing number of studies are now emerging that provide mechanistic insights substantiating the hypothesis that there is a novel, noninfectious mechanism through which the spike protein of SARS-CoV-2 can bind endothelial cells by interacting with different host receptors, leading to multiple instances of endothelial injury (Perico, Benigni & Remuzzi 2023).*

You know what else the endothelium does? It serves as the blood-tissue barrier (such as the blood-brain barrier) for various organs. Endothelial damage by a potent cytotoxic agent or pathogen in the blood will also be more likely in the narrower blood vessels such as capillaries, where blood flow is slower, and where a larger percentage of the vascular fluid is in immediate contact with the endothelium. This can also be seen in case studies where cytotoxic agents have been injected into the blood, where the first capillary beds encountered exhibit the earliest signs of significant damage (Domej et al. 2007).

Endothelial damage will by extension also be more likely in the tight junctions that form the key structures of the blood-tissue barriers, where, as we previously observed, the LNPs can intiate transfection and exert cytotoxic effects. And according to Perico, Benigni & Remuzzi, the spike protein can also cause endothelial injury independently.

Penetration of these barriers are bad enough, and that by itself has the potential to cause numerous significant adverse events, ranging from neurodegenerative diseases to sepsis, autoimmune processes, hormonal imbalances, fertility issues, mental disorders and even the exacerbation of cancer.

What's more, however, is that transfection by the LNP in the epithelium of e.g. the blood-brain barrier is also going to have spike protein penetrate into the protected tissues.

The potential consequences of a large-scale introduction of spike protein are an obvious explanation for the unprecedented number of adverse neurological events being reported in

pharmacovigilance databases such as VAERS in connection to the covid vaccines. More specific causal mechanisms abound. A recent article by Martinez-Mármol (2023) discuss how spike causes progressive fusions between neurons and between neurons and glia in the brain, “severely compromising neuronal activity”, potentially giving rise to a whole host of pathologies, but not least ones similar to those behind Lewy-body dementia:

> *Tunneling nanotubes are similar cellular bridges that allow communication between cells and have been reported to mediate the transport of toxic α-synuclein aggregates. Our results demonstrate the formation of neuronal bridges that can extend hundreds of micrometers, allowing the exchange of small proteins and large mitochondria between interconnected neurons (ibid).*

Overall, the broadly problematic effects of the presence of spike protein in our bodies have been explored in some detail, not least since they could plausibly be connected to the perils of the SARS-CoV-2 pathogen (but again, spike distibution after infection is relatively limited as opposed to after mRNA inoculation on the LNP platform).

An interesting study on zebrafish (their ACE2-receptors are similar to ours) from last year shows that spike protein is toxic to marine animals (Ventura Fernandez et al. 2022). The authors even emphasize the potential ecological harm from contaminated wastewater, and the damage seen in the animals is wide-ranging:

> *We demonstrated, for the first time, that zebrafish injected with fragment 16 to 165 (rSpike), which corresponds to the N-terminal portion of the protein, presented mortalities and adverse effects on liver, kidney, ovary and brain tissues.*
>
> *…*
>
> *The application of spike in zebrafish's olfactory epithelium causes thrombosis of the deep medullary veins. Damage to the structure and function of this system can lead to severe encephalitis, toxic encephalopathy, and, after viral infections, severe acute demyelinating lesions (ibid).*

The main problematic mechanism of action of the spike protein, generally speaking, seems to be that it elicits inflammation and cellular abnormalities through triggering certain internal signaling functions of the affected cells. This was observed by Suzuki & Gychka in 2021, and was elaborated on by Tyralska et al. (2022) where the inflammatory response was connected to tissues where the ACE2 receptor is prominent, such as the endothelium.

Spike protein also, through this signal triggering, seems to play a direct and independent role in the emergence of clotting disorders, thrombosis, pulmonary damage and neurogenerative disorders (Letarov, Babenko & Kulikov 2020) by promoting the formation of amyloid plaques independently of the prion-related pathways discussed above (Idrees & Kumar 2021).

Jiang & Mei (2021) also showed that spike protein, by another separate molecular mechanism, inhibits cellular processes for repairing DNA damage. This is a significant red flag, since these intracellular DNA repair mechanisms are one of the key factors in preventing the runaway mutations that cause cancer. Incidentally, both of the proteins Jiang & Mei identified as being suppressed by the spike protein are connected to breast and ovarian cancer in particular (these cancers emerge when said proteins are underexpressed), so here's a very clear-cut explanation for important parts of epidemiological data.

To be as explicit as possible - the disturbance of DNA repair mechanisms is one of the central causal factors behind cancer, cellular aging, and a long list of catastrophic syndromes. That the spike protein "significantly" disturbs DNA repair mechanisms in general, is very much a cause for alarm.

Aberrant DNA repair mechanisms have also recently been implicated in the emergence of autoimmunity (Manolakou, Verginis & Boumpas 2021).

mRNA-related risks

There are also specific pathologies related to the mRNA component of the LNP, i.e. the payload that then translates into spike protein production in the ribosomes. Zhang et al. (2021) showed that the mRNA can "reverse transcribe" into human DNA in vitro, and thus become expressed in human tissue, i.e. the mRNA can enter into the genome of human cells and affect their future development, which was followed up by Aldén et al. (2022), showing reverse transcription taking place in human liver cells in vitro. Jessica Rose published a follow-up comment on these issues just yesterday, and argues that we need to do further studies sampling human DNA and ascertain whether or not these reverse transcription events commonly take place in vivo, i.e. in living human subjects.

Immunological dysregulation, priming and "leaky" vaccines

The priming and dysregulation problems are issues we've been talking about since around November-December 2022, and which were covered in part by our conference in Stockholm in January this year. These are independent of the risks connected to localized inflammation and rather relate to the inoculations' "programming" of our immune system. The priming and dysregulation problems are possibly the most disturbing consequences of the mRNA vaccines, but we still haven't got a very clear picture of the overall implications.

There's also a related issue with the effects of incomplete immunization for virus evolution and how this has influenced the character of later covid variants.

Immunological suppression and IgG4 promotion

To begin with, the mRNA covid vaccines generate a certain form of immune system suppression and dysregulation. Late last year, research by Goh et al. (2022) and Irrgang et al.

(2023) provided evidence for how the vaccinations, especially the boosters, alter the immune profile of recipients such that covid infections get "tolerated" by the immune system.

Basically, there's a substitution in the vaccinated of virus-neutralizing antibodies for non-inflammatory ones, a "class switch" from antibodies that work towards clearing the virus from our system, to a category of antibodies (IgG4), one of whose purposes is to desensitize us to irritants and allergens. One of their important roles is to render you "immune" to allergens, so that they do not trigger an unneccessary inflammatory response.

When this happens in relation to a viral pathogen like SARS-CoV-2, the unhelpful result is rather that the virus can remain in the body and its tissues and keep replicating.

This substitution towards IgG4 is extensive, meaning that its particular subcategory of antibodies gets dominated by the non-inflammatory (rather than virus-clearing) type.

Follow-up research by Uversky et al. (2023) gives us a straightforward description of the problem complex:

> *Additionally, recent investigations have found abnormally high levels of IgG4 in people who were administered two or more injections of the mRNA vaccines. HIV, Malaria, and Pertussis vaccines have also been reported to induce higher-than-normal IgG4 synthesis.*
>
> *…*
>
> *However, emerging evidence suggests that the reported increase in IgG4 levels detected after repeated vaccination with the mRNA vaccines may not be a protective mechanism; rather, it constitutes an immune tolerance mechanism to the spike protein that could promote unopposed SARS-CoV2 infection and replication by suppressing natural antiviral responses.*
>
> *Increased IgG4 synthesis due to repeated mRNA vaccination with high antigen concentrations may also cause autoimmune diseases, and promote cancer growth and autoimmune myocarditis in susceptible individuals (ibid).*
>
> *…*
>
> ***More IgG4 seems to be linked to more aggressive cancer growth, and both were strongly associated with higher cancer malignancy and poor prognosis.***

In terms of immunity suppression, the net effect is that the inflammatory response to covid infection gets down-regulated. Full-blown infections will present with milder symptoms, and they won't get cleared as effectively.

We also know that vaccination induces quite a different immune response in comparison to a covid infection. In other words, if you are vaccinated before exposure to the virus, the tolerance-inducing IgG4 class switch will manifest more strongly. If you on the other hand were exposed to the virus before inoculation, the class switch is less prominent (Kizsel et al. 2023).

In relation to all of the above, it's therefore plausible that multiply vaccinated individuals will tend towards a situation of long-term, repeat infections that do not get cleared, and which promote systemic damage.

As I wrote last year, combine this with the extensive data on OAS/the Hoskins effect, and we potentially get two separate avenues for immune suppression. I.e. the first one would be the Hoskins effect/antigenic original sin where immunity fixates on the narrow vaccination, the SP from the classic "Wuhan strain" (rather than to the 29 proteins of the entire virus, which would engender a more robust immunity that minor mutations in the virus could not easily get around), and the second one would be in terms of this IgG4 substitution.

Both of these separate avenues could then independently undermine the capability of the multiply vaccinated to clear not only covid infections but increasingly also other viral infections which may opportunistically evolve to exploit this immunity gap created by substitution and an overabundance of antibodies that do not clear viruses. This then potentially results in these "invisible", low-intensity infections which elicit a weak inflammatory response - yet which crowd the body with viruses and promote systemic damage.

This situation manifests with a whole set of potential long-term complications of its own.

IgG4-related disease

IgG4 dysregulation as such has a number of further related issues as well. IgG4-related disease is associated with high serum levels of IgG4 (or consequent collections of material in the body rich in IgG4-positive white blood cells) (Khosroshahi & Stone 2011), which is exactly what the mRNA vaccines evidently induce.

IgG4-related disease is connected to a whole host of related diseases, all of which involve inflammation, scarring, and tissue-destroying formations of masses of connective tissue in various parts of the body. These processes are associated with a whole host of negative outcomes. Almost all organs can be affected through blocking the vascular system and inflammation, and there's a long list of potential neurological and cardiovascular issues arising from this, not least aortits, thrombosis and pericarditis. Inflammations of the arteries and the aorta are the most common cardiovascular manifestations (Koo, Lim & Chan 2021).

There's also a connection between IgG4RD and autoimmune disorders (Maslinska, Dmowska-Chalaba & Jakubaszek 2022).

The potential association between inflammatory fibrosis and the amyloid formation is clear enough, and the possible synergies with the two other separate pathways towards amyloidosis in the spike and through the aberrant peptide production discussed above, should not be disregarded.

Finally, the persistence of low-grade inflammation facilitated by the tolerance effect of the class switch and the continuous or repeated presence of the covid infection (as evidenced by the record wastewater measurements seen all over the world) is inevitably going to have measurable long-term consequences in terms of everything from cancer promotion to accelerated cellular aging.

Conclusions

There are so many modalities of possible harm here that it's almost preposterous.

We're not just talking about a couple of elevated risks, but a whole plethora of significant causal mechanisms, with several, potentially synergistic factors behind almost every single potential negative health outcome that can be discerned in the research.

And paradoxically, this overwhelming set of indications is part of the problem. It immediately generates cognitive dissonance. Because on the face of it, it's not reasonable to entertain the idea that an ostensibly beneficial pharmaceutical product that almost everyone agrees has saved millions of lives, is associated with such an extensive set of etiological mechanisms.

In other words, we're getting into the territory of the psychology of the "big lie" here.

The epistemic conditions are such that the very abundance of evidence makes it very difficult to accept the conclusion. Not least given the Nobel award, the situation is one where the average person, without delving into the data, and without a strong, epistemic anchoring of his or her own, would generally be *less rational* to accept the conclusion indicated by the evidence.

And this is precisely why I think we now need to compile these comprehensive overviews of etiological mechanisms, and connect them to the epidemiological data as clearly as possible, to provide a stable and accessible foundation for the proper assessment of the effects of these products.

William M Briggs - if you're reading this, perhaps you could be persuaded to give us a follow-up overview pertaining to the possible questions we could ask ourselves regarding connections between these mechanisms and the epidemiological data.

Instead, however, we dole out the Nobel Prize in support of big pharma's marketing campaigns. We awarded the Nobel Prize in medicine for a product that certainly did vastly

more harm than good - something which was obvious and easy to ascertain more than two years ago.

Would it really have been so difficult for the committee to perhaps research one or two of these modalities for potential harm before deciding to place their royal acclamation on an obviously unsafe experimental therapy whose long-term consequences are just beginning to show?

All of this should obviously have been stopped as soon as we had minute indications of serious adverse events in the pharmacovigilance data, and the presence of several plausible mechanisms of harm should be the end of the discussion.

But perhaps they really are that incompetent.

Perhaps Rintrah's outlook is in this case correct. That these are not serious people, that "it's like they're not really trying to think, but rather, they're trying to gather the words they like and then they try to find some sort of rhetorical string to tie them all together with."

And journalists. All the papers are now yellow papers, it seems. Journalists are the group of people tasked with doing this sort of investigative work and sticking their necks out. Instead, we find them uncritially fawning over the Nobel Prize recipients, musing on the crucial role played by Xerox machines in bringing these brilliant geniuses together so that countless millions of lives could be saved, as they lambast actual, critical research as "conspiracy theory".

And all the while, excess deaths remain high all over the world throughout 2023.

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