An Independent Biosecurity Risk Assessment on the Current Experimental Mass Vaccination Campaign

Jonathan Weissman BSc MSc MSc
Northern Ireland, UK
JWeissman123@outlook.com
alltherisks.com
17 September 2021

Disclaimer: I have written this document in a completely personal capacity. I am unaffiliated with any political movement and declare that I have no financial or other competing interests. My part-time biosecurity research is completely independent of my full-time career in cybersecurity. This document is written as a biosecurity risk assessment to inform the general public of the ongoing risks associated with the current mass vaccination campaign using experimental gene therapy vaccines to target the spike protein of the original SARS-CoV-2 viral strain first identified in Wuhan. This is a working document that may contain mistakes since it has not been reviewed by anyone prior to its release and is being shared in reaction to the UK government's increasing attempts to mass vaccinate schoolchildren. Please email me if you find any errors, indicating whether you would like to be acknowledged in subsequent revisions.

The Lord is my light and my salvation; whom shall I fear? the Lord is the strength of my life; of whom shall I be afraid?

When the wicked, even mine enemies and my foes, came upon me to eat up my flesh, they stumbled and fell.

Though an host should encamp against me, my heart shall not fear: though war should rise against me, in this will I be confident.

One thing have I desired of the Lord, that will I seek after; that I may dwell in the house of the Lord all the days of my life, to behold the beauty of the Lord, and to enquire in his temple.

Psalm 27:1-4

And he said to them all, If any man will come after me, let him deny himself, and take up his cross daily, and follow me.

For whosoever will save his life shall lose it: but whosoever will lose his life for my sake, the same shall save it.

For what is a man advantaged, if he gain the whole world, and lose himself, or be cast away?

Luke 9:23-25

And Jesus answered them, saying, The hour is come, that the Son of man should be glorified.

Verily, verily, I say unto you, Except a corn of wheat fall into the ground and die, it abideth alone: but if it die, it bringeth forth much fruit.

He that loveth his life shall lose it; and he that hateth his life in this world shall keep it unto life eternal.

If any man serve me, let him follow me; and where I am, there shall also my servant be: if any man serve me, him will my Father honour.

John 12:23-26

Executive Summary

- Risk of anaphylaxis and severe adverse reactions to polyethylene glycol (PEG), an additive not used in vaccines before
- Risk of acute fatal thrombotic blood clotting events
- Risk of reactivation of latent viruses, including the virus that causes shingles
- Risk of inflammation of the heart muscle, myocardium, and risk of inflammation of the pericardium, a protective fluid-filled sac surrounding the heart
- Risk of development of fatal disease pulmonary arterial hypertension
- Risk of development of cardiovascular diseases including coronary artery disease, systemic hypertension and stroke
- Risk of LNP-induced cytotoxic activity from mRNA vaccines damaging organs
- Risk of novel cationic/ionisable lipids inducing cytotoxic activity
- Risk of vaccine-induced spike proteins rupturing the blood-brain barrier (BBB), allowing LNPs and spike proteins to cross the BBB leading to neurological damage
- Risk of development of Guillain-Barre syndrome (GBS) and other nervous disorders
- Risk of development of fatal neurodegenerative prion disease due to spike protein misfolding
- Risk of damage to the lining of blood vessels due to freely circulating vaccine-induced spike proteins
- Risk of hypertension, increase in blood pressure in blood vessels
- Risk of organ damage due to indiscriminate distribution of LNPs or viral particles and vaccine-induced spike proteins
- Risk of damage to female reproductive tissue and infertility due to molecular mimicry
- Risk of dramatic increase in autoimmune disease due to cross-reactive antibodies
- Risk of microthrombotic blood clotting events
- Risk of vaccinal shedding, where vaccinees expel biologically active material causing adverse reactions in others
- Risk of antibody dependent enhancement (ADE), where vaccinal antibodies increase the ability of the virus to infect cells, potentially in low or waning antibody levels
- Risk of vaccine-enhanced disease worsening disease outcomes after contracting the virus
- Risk of impaired ability to fight common-cold-causing coronaviruses
- Risk of mass vaccination campaign exerting immune-selection pressure to proliferate dominance of immune-escape variants, such that vaccinal antibodies no longer neutralize the virus
- Risk of proliferation of increasingly infectious variants due to conducting mass vaccination campaign with non-sterilising vaccines during a pandemic on a background of high infectious pressure
- Risk of original antigenic sin (OAS), where attempts to retrain the immune response through revaccination fail following immune-escape spike protein mutations, freezing the original outdated immune response acquired in the initial vaccination
- Risk of catastrophic increase in morbidity and mortality due to aggressive mass vaccination campaign leading to emergence of dominant highly infectious and immune-escape variants, coupled with OAS
- Risk of catastrophic Catch 22 situation: vaccinees may require revaccination due to ADE in waning antibody levels but revaccination with updated spike protein may recall initial antibody response due to OAS, further suppressing their innate immunity

- Risk of tragic and unnecessary deaths if proven early-intervention treatment protocols, using antiviral medication and vitamins, continue to be deliberately suppressed by public health bodies and government agencies
- **Risk of devastating increase in all-cause morbidity and all-cause mortality**
- **Risk of societal breakdown as a direct result of deliberate mismarketing of safety and efficacy clinical trial data, deliberate understatement of potential pathological risks of vaccination, deliberate overstatement of efficacy benefits, immoral use of coercion, suppression of early intervention treatments, suppression of warnings from distinguished researchers and academics on safety and efficacy, and deliberate ignoring of safety signals in public adverse reaction warning systems**

All the risks listed in the executive summary are described in this risk assessment. Through citing academic experts in internationally recognised peer-reviewed journals or recent preprints, actual pathologies and epidemiological phenomena underpinning these risks are provided. The risks vary in scope from the individual to the entire population. The risks also vary in severity. Most are at least severe, whilst some are critical, some potentially threatening the majority of the population. None of these risks can be discounted at this stage since all the gene therapy vaccines deployed are experimental, with the novel mRNA vaccines in clinical trials until 2022 (Moderna)[1] and 2023 (Pfizer).[2] The scientific fields of toxicology, molecular biology, virology, immunology and epidemiology are all invoked to outline these risks in appropriate detail. Any attempt to intervene in a pandemic of a novel RNA virus without sober and careful consultation of all these specialist scientific areas is futile, reckless and dangerous.

All attempts to silence those warning of the foolhardiness of this strategy are reprehensible, to some it may even seem to be unforgivable. The use of social engineering and coercion to influence people to enlist against their will in an unprecedented genetic experiment is immoral to the point of criminal. Targeting children in the very same manner is yet more morally outrageous.

Arbitrary and punitive laws have already been imposed without rigorous scientific evidence or logical reasoning, in the absence of moral clarity to justify such unprecedented measures. Although in recent times personal responsibility has mostly become subservient to absolute civic obedience that asks no questions of state-sponsored and state-broadcast scientific dogma and no longer holds policymakers to account for their failures, in times as unprecedented as these, some will always dare to question the status quo without fear of reprisal in whatever form, motivated by their love of God, their love for their fellow man and their passion for true science. Secure in the moral framework that guides them, unfazed by their own mortality, unwavering in their commitment to do good and to shun evil irrespective of the cost, such people will not and cannot be dissuaded by cowardly detractors, who hold power and would think nothing of unjustly wielding it against them simply because they cannot allow their own immature and short-sighted arguments to be exposed in the democratic arena of open public debate. The determination of such innovative thinkers to raise their voices above the baying mob, and overturn an entrenched but wholly incorrect consensus, has characterised great scientists of the past, who are celebrated by all today.

The stakes could not be higher. Rates of severe morbidity and mortality now threaten our society far beyond anything experienced in living memory. This in turn threatens the social order. Beyond even this, the influence of pan-national organisations and individuals of great prominence and influence stand ready to capitalise and may yet force upon us an even more acute inflection point in the fragile and unfolding story of humanity. Reengineering the essential human fabric that unites humanity with even greater audacity than achieved by the current class of experimental gene therapies may invigorate some, but it repulses others beyond measure. Reimagining global society according to the blueprint of immensely powerful, ideologically driven stakeholders who seek to bind humanity in a total global collective is thus recognised as an unacceptable pivot for some, who now find themselves compelled to speak out.

I now urge you, my reader, to cautiously consider the content within this document and to verify for *yourself* whether this document is accurate and even prescient.

Jonathan

Table of Contents

Introduction

As a cybersecurity professional, I appreciate the importance of third-party risk management, which is the management of risks an entity is exposed to by virtue of relationships forged with external parties. With this in mind, I have surveyed academic journal articles, experimental laboratory data, clinical reporting data, regulatory documents, and real-world safety and efficacy data from governments and public health bodies. My goal in so doing is to verify the credibility of the claims made by vaccine manufacturers, regulatory bodies, governments and public health bodies with respect to safety, efficacy, ethicality and strategic viability concerning the experimental medical interventions marketed as COVID-19 vaccines. I believe that objective (unbiased) and thorough scrutiny of the current public health strategy forces us as a society to reconcile with the immediate halting of this experiment and strategic diversification in tackling SARS-CoV-2 (COVID-19).

Novel medical advances can only ever be *safely* and *ethically* adopted by a population sufficiently informed by their medical leadership of the known safety and efficacy profiles of the medical intervention at hand, with clear transparency where risks remain unknown. The intervention must be properly moderated by impartial adjudicators (i.e., regulatory bodies), and the general public must be able to hold them to account if they fail in their duties. A medical technocracy holding so much power with a compelling narrative (i.e., a theory) so untouchable that it cannot even be questioned is fundamentally *incompatible* with genuine public health risk management.

Freedom for scientific debate is a prerequisite for scientific advancement and any human enlightenment it offers. The absence of it is a danger to us all. Under the guise of public health risk management, to prevent the spread of SARS-CoV-2, wedding days have been cancelled, schools closed and millions isolated from loved ones at times of need. Highly disturbing recent research even suggests that children's verbal, motor and overall cognitive performance is significantly declining as a result of the pandemic.[3] It is surely then logically inconsistent for the population to be subject to such extreme infection control measures (i.e., lockdowns) and yet for the experimental gene-therapy mass vaccination programme to proceed without due scrutiny of risk at the individual and population level. According to the Department of Health and Social Care, it is "unfair" to expect healthcare professionals or vaccine manufacturers to "take responsibility for the consequences of the use of that medicine in the way that they normally would."[4]

All three vaccines authorised for temporary use under Regulation 174 by the MHRA in the UK are gene-therapy based. This novel vaccine technique delivers mRNA (Pfizer/Moderna) or DNA (AstraZeneca) genetic code to instruct cells to first create the spike protein antigen. In turn, this induces a specific anti-spike antibody response. Such gene therapies were touted as being advantageous over traditional live attenuated vaccines (LAVs), which could have offered broader protection,[5] due to how easily and quickly the genetic code can be updated and the vaccine redeployed. However, to this day, the first-generation vaccines deployed still include the genetic code for the *original* SARS-CoV-2 strain first identified in Wuhan, despite subsequent mutations in the spike protein characterising the currently dominating Delta variant. There is already an appreciable incidence of breakthrough infections amongst vaccinees with the Delta variant,[6,7] with infected vaccinees experiencing equivalent viral loads to infected unvaccinated individuals.[8–10] Prior to the CDC suspending their reporting on breakthrough cases, according to

their own analysis of preliminary data on breakthrough infections to April 30, 2021, 10% of breakthrough infections led to hospitalisations and 2% led to death.[7]

No vaccine has ever been approved to protect against disease caused by any of the six prior coronaviruses.[11] Furthermore, no mRNA or DNA vaccine has previously been approved.[12] Vaccine development takes an average of 10 to 15 years.[13] The safety profile of mRNA vaccines in humans is unknown since no large trial on their usage has ever previously been completed. The only safety data on their usage are the Phase I and Phase II clinical trials of the first-generation SARS-CoV-2 genetic vaccines[14] and their ongoing Phase III clinical trials. With respect to their Phase III trials, Moderna have confirmed **"**[a]s of April 13, all placebo participants have been offered the Moderna COVID-19 Vaccine and 98% of those have received the vaccine."[15] Pfizer's follow-up safety study lasted for 6 months after the second injection, consisting of both blinded and open-label periods. In the clinical trials, the pharmaceutical companies computed how much less likely the vaccinated group were to contract SARS-CoV-2 as compared to the control group to supposedly demonstrate their products' efficacy.[16,17] These headline relative risk reduction (RRR) figures of approximately 95% for Pfizer and Moderna have been used to market the products to the general public. However, absolute risk reduction (ARR), the raw disease risk difference between the vaccinated and placebo groups, is just 0.7% and 1.1% for Pfizer and Moderna respectively.[18] This has been suggested as an example of outcome reporting bias, "which ignores unfavorable outcomes and misleads the public's impression and scientific understanding of a treatment's efficacy and benefits."[18] Recent real-world data from the Israeli government suggests that RRR against the Delta variant is now just 39%.[6] Furthermore, following Dr Bart Classen's statistical analysis of the publicly available clinical trial data, when weighting severe vaccine-induced adverse events the same as disease outcomes of equivalent severity resulting from SARS-CoV-2 infections, all-cause severe morbidity in the Phase III clinical trials was statistically significantly higher in the Pfizer and Moderna vaccinated groups compared to the placebo groups.[19] In the Pfizer trial, there were 262 severe events in the vaccinated group and 172 in the control group, whilst 3985 severe events occurred in Moderna's vaccinated group with 943 in their control group.[16,17,19] There no longer remains a control (placebo) group to understand the impact of either of the two experimental mRNA-vaccines currently available to the public, despite the novelty of deploying mRNA vaccine technology, let alone at global scale in the midst of a pandemic.

Background to RNA Viruses

The field of epidemiology has long since recognised RNA viruses as being especially difficult to confidently control through human intervention,[20,21] especially should an acutely pathogenic RNA virus emerge. The literature identifies their distinctively high mutation rate[22] as compared to DNA viruses.[21] It is, of course, an oversimplification of the mechanism of the evolution of RNA viruses to merely claim that their higher rate of mutations implies a faster rate of viral adaptation.[23] This is because the overwhelming majority of mutations are unfavourable and hinder adaptation.[23] Unprecedented human intervention can, though, artificially shape the course of a pandemic of a novel RNA virus in equally unprecedented ways. In the literature, the dynamic population of genomic mutant strains comprising an RNA virus is collectively known as the *quasispecies*.[24,25] Even within a given infected host, multiple distinct strains of an RNA virus may simultaneously occur and even amalgamate in a recombination event.[26] The challenges of controlling an RNA virus through antiviral drugs is well-documented, due to their propensity to acquire antiviral resistance mutations.[27] For this reason, multi-drug interventions are preferred for treating HIV infections.

Immune-selection pressure exerted by vaccinal antibodies narrowly focussed on the SARS-CoV-2 spike protein likely provides a classic example of positive selection, whereby antigenic variants that circumvent the acquired (vaccinal) host immune response are positively selected and thus proliferate both within the host and, ultimately, within the quasispecies distribution of the virus at the population level. Positive selection is, generally speaking, a key driver in the long-term evolution of an RNA virus.[28] Viruses with such genetically-diverse quasispecies populations are very resilient to challenges in the host environment, precisely because there may always be a variant amongst the quasispecies that can overcome a novel environmental challenge, and ultimately thrive and dominate.[25] As far back as 2010, researchers identified quasispecies theory and the interpretation of real-time genomic sequencing data as key to predicting the evolution of an RNA virus.[24] Microbiologists have even already sequenced the quasispecies for a single patient hospitalised with SARS-CoV-2, even though they only developed a "mild" form of the disease.[29] Even prior to mass vaccination, the S-gene, encoding for the spike protein on the virus' surface, was shown to have the highest density of (nonsynonymous) variants amongst four of the main SARS-CoV-2 genes: ORF1a, ORF1b, S and N.[30] Under simultaneous global (often suboptimal) evolutionary pressure exerted by vaccinees with a highly specific anti-spike antibody response, the viral spike protein is thus increasingly liable to mutate to evade an increasingly hostile host environment.

Vaccine Safety

Aside from alarming unanswered questions on whether novel mRNA technology can insert into human DNA and cause potentially harmful mutations that proliferate to the next generation,[31] there is an urgent need to understand whether the vaccinal spike protein is well-tolerated by the body. The spike protein itself is comprised of an S1 subunit containing the receptor-binding domain (RBD) and an S2 subunit responsible for the cell membrane fusion reaction. The SARS-CoV-2 spike protein alone is known to inflame the vascular endothelial cells that line blood vessels.[32] Damaged endothelial cells can cause thrombosis and hypertension.[33] Experimentation on mice has demonstrated that the virus can cross the blood-brain barrier (BBB),[34] whilst advanced in vitro models of the human BBB also show that the S1 subunit degrades barrier integrity, likely via directly inducing pro-inflammatory responses in brain microvascular endothelial cells (BMECs).[35] Damage to BMECs compromises the integrity and permeability of BMEC, increasing indiscriminate permeability of the BBB,[36] further increasing the risk of freely circulating lipid nanoparticles (LNPs), full spike proteins and S1 subunits crossing the BBB following injection. This could potentially disrupt the delicate neural networks within the brain.[37–40] Post-mortem data has already shown that the virus can cross the human BBB.[41]

In the disease caused by SARS-CoV-2 infections, the RBD of the spike protein on the virus' surface binds to angiotensin-converting enzyme 2 (ACE2), a protein expressed on the surface of cells in tissues throughout the body,[42] which acts as a receptor to permit the attached SARS-CoV-2 virus to invade and infect cells through a membrane fusion reaction at the cell surface. Aside from spike-to-endothelial-cell binding due to RBD-to-ACE2 affinity, recent in vitro experimentation suggests that the spike protein also downregulates the expression of critical endothelial junctional proteins, decreasing barrier integrity and so increasing barrier permeability.[43] Proteins VE-Cadherin, PECAM-1, JAM-A and Connexin-43 contribute to the immune response and wound healing and so their downregulation could induce cardiovascular damage.[43] Each of the experimental gene-therapy vaccines offered in the UK encode for the full SARS-CoV-2 spike protein, with the Moderna and Pfizer product BNT162b2 including the two deliberate "2P" proline substitutions K986P and V987P in the S2 subunit.[44,45] These mutations were previously publicised in 2017 with respect to the spike protein of MERS-CoV, ostensibly with the aim to maintain the spike protein's prefusion conformation[46] to preserve epitopes that promote antibody neutralisation[47] and, crucially, to avoid shedding the S1 subunit.[48] However, generally, RBD-to-ACE2 binding destabilises the prefusion spike trimer leading to S1 shedding.[49] Moderna and Pfizer simply reapplied these prior known "2P" mutations during development in 2020. Where is their evidence of safety and stability in vivo with respect to SARS-CoV-2?

Despite the theory of the vaccine manufacturers, a study in the Clinical Infectious Diseases journal demonstrated that the S1 subunit was found at detectable levels circulating in the plasma of 11 out of 13 healthcare workers who received the experimental Moderna injection.[50] Importantly, the authors were able to rule out SARS-CoV-2 infection as the source of the freely circulating S1, on account of insignificant concentrations of nucleocapsid, one of the four structural proteins of the virus.[50] They suggested two mechanisms for this: circulating protease enzymes breaking the peptide bonds that exist between the S1 and S2 subunits and the immune response (i.e., cytotoxic T cells) killing spike-protein-expressing cells, releasing spike proteins directly into the bloodstream.[50] Their results showed appreciably higher levels of the S1 subunit relative to the full spike protein.[50] Freely circulating S1 and the full spike protein both represent the pathological risk previously discussed since S1 alone contains the RBD. The study authors

conclude an association relationship exists between an IgG and IgA antibody response elicited by the immune system and the removal of both S1 and the full spike protein in circulation.[50] IgG and IgA were initially detected at 5 days post-injection,[50] thereby inferring it is very common for RBD-containing S1 or spike protein antigens to circulate (and hence act pathogenically) without significant antibody challenge for at least 5 days following injection. Following the first mRNA vaccine dose, it typically takes 18-21 days for IgG levels to fully mature and plateau.[51] Biochemist researchers recently hypothesised "substantial" shedding of S1 from virions and naturally infected cells.[52] Furthermore, in vitro experimentation demonstrates that the spike protein directly activates the complement pathway, which could help to explain disease manifestations including thrombocytopenia.[53] It is incumbent on manufacturers to provide credible evidence to support their claims with respect to the safety and stability of the vaccinal spike protein.

The phenomenon whereby viruses and bacteria replicate proteins in the host to disrupt its *cellular signalling* mechanism to enhance the survival odds of the pathogen, is known and documented in the literature.[54] Since the goal of a virus is to survive and replicate within the host (and to infect new hosts), circumventing the host's innate and acquired immune response by interfering with (or leveraging) the host's cellular signalling mechanisms can be advantageous. The ACE2 enzyme acts as a catalysis in the hydrolysis reaction that converts peptide hormone angiotensin II (Ang II) into Ang(1–7). However, the spike protein interferes with this physiological function of ACE2, as the spike protein's RBD binds with the ACE2 enzyme as a membrane receptor on the cell surface. The impact of this binding is that Ang II levels increase, which is known to increase blood pressure and cause hypertension.[55,56] The potential for this vaccine-induced pathogenesis to cause pulmonary arterial hypertension (PAH), a presently incurable and often undiagnosed disease with an exceptionally high fatality rate with or without treatment, has already been suggested in an article in the journal Vaccines.[57] Aside from pulmonary endothelial cells, cardiovascular cells (also expressing the ACE2 gene) could also be affected in this way, which could cause cardiovascular diseases including coronary artery disease, systemic hypertension and stroke.[57] Furthermore, toxic overabundance of Ang II increases the production of reactive oxygen species (ROS) that may act pathogenically, whilst decreasing Ang(1-7) peptide synthesis, itself important in preventing oxidative-stress-induced cellular damage.[58]

Aside from freely circulating spike protein, the LNP-encapsulated mRNA formulation also distributes in the body. Publicly disclosed data from the Japanese government health regulator, PDMA, shows that Pfizer's LNPs distributed across the body of the rat test subjects.[59] As is common practice, this study genetically encoded the firefly protein luciferase instead of the SARS-CoV-2 spike protein.[59] 48 hours post-injection, 16%, 1% and 0.1% of the formulation concentrated in the liver, spleen and ovaries respectively.[59] The study also records the detectable gene expression in liver cells, as the organ synthesised the luciferase protein and emitted light.[59] This raises further immediate and urgent safety concerns. In the absence of other public data provided by pharmaceutical manufacturers on bio-distribution in animal test subjects for the cellular expression of spike protein to assuage safety concerns, the little pharmacokinetics data publicly available is alarming. Indeed, as early as 2015, it was known that intramuscular administration of mRNA-LNPs in mice led to the systemic spread of LNPs and widespread protein expression.[60] The Japanese PDMA biodistribution data simply reaffirmed such pre-existing concerns that were not made publicly available by the MHRA prior to the UK vaccination programme starting in December 2020. Thus, even prior to the experimental vaccination campaign, it could be expected that not all of the mRNA formulation would be taken up by macrophages and dendritic cells at the deltoid muscle nor via draining lymph nodes. Instead, following the PDMA data, the increasing detection of the radioactively labelled mRNA

LNP formulation in the plasma and liver suggests it increasingly circulated via the circulatory and lymphatic systems in the 2 hours following injection. These two biological transport systems can explain the peak concentrations of the LNPs observed in various organs at different time points in the 48 hours following injection.

From the study on rats, the diffusion of the LNPs in the human body can be presumed to also lead to local cellular expression of the SARS-CoV-2 vaccinal spike protein. Since tissues throughout the body express the ACE2 gene, there is yet further reason to believe that pathological disruption to the body's cellular signalling mechanism occurs in different organs. A recent paper published in the European Journal of Internal Medicine on this risk of vaccination concludes that the "resulting pathological features may resemble those of active coronavirus disease".[61] Spike proteins synthesised by cells migrate from the cytoplasm to the cell's surface. The cell, thus resembling a SARS-CoV-2 infected cell, may be targeted and destroyed by the immune system via cytotoxic T-cell activation or may die first. In either scenario of cell death (apoptosis), this same paper describes the potential for this to result in free-floating spike protein debris, which again can lead to the pathological downregulation of ACE2 and subsequent inhibition of Ang II conversion, as already described.[61] It is generally known that the heart's cytotoxic T-cell immune response can permanently damage heart tissue, impair cardiac function and lead to lethal acute or chronic heart failure. A presentation by John R. Su of the CDC COVID-19 Vaccine Task Force, analysing VAERS case reports following vaccination in the U.S. up until 18 August 2021 is publicly available on the CDC website.[62] It identifies 671 reports of pericarditis – inflammation of the pericardium, a protective fluid-filled sac surrounding the heart – and 1,903 reports of myopericarditis, a complication of acute pericarditis.[62] A recent preprint using VAERS data suggests that the probability of a male vaccinee aged 12-17 experiencing a cardiac adverse event following their second Pfizer or Moderna injection is higher than their 120-day hospitalisation risk due to contracting SARS-CoV-2.[63] Aside from acute myocardial inflammation and other acute cardiac adverse reactions, how much other vaccine-induced heart damage is silently occurring?

Given the PDMA rat pharmacokinetics data, applying the precautionary principle,[64] it must be assumed that the LNPs distribute across the human body too. Following intramuscular injection, LNPs not taken up by antigen presenting cells (APCs) locally recruited at the injection site[65] in the deltoid muscle may drain via the lymphatic system[66] and distribute in the bloodstream before settling across the body, where they are then taken up by distant cells via endocytosis, leading to the genetic expression of spike proteins that migrate to cell surfaces, which finally induces cytotoxic-T-cell-mediated apoptosis and the release of spike proteins. Besides even this, it has previously been shown that LNPs can enter the brain easily.[31] The indiscriminate distribution of the LNP mRNA formulation across the body, coupled with the indiscriminate cellular uptake via endocytosis, the biological process by which cells internalise extracellular plasma membrane enriched with proteins and lipids,[67] means that damage could be caused widely across the body. Freely circulating spike proteins, S1 and LNPs hence represent potential vectors to attack ACE2-gene-expressing[68] lymphatic and vascular endothelial cells, which form an interface between circulating blood and tissues.[69] Other important questions with respect to the biological mechanisms of LNP-mediated mRNA gene delivery remain unanswered in the literature, such as the fate of both the RNA and the other LNP components once inside cells.[70] The SARS-CoV-2 viral spike protein can also bind directly to ACE2-expressing platelet cells and enhance platelet activation, leading to both pro-inflammatory cytokine responses and the formation of leukocyte-platelet aggregates (LPAs).[71] LNPs distributing through plasma can thus induce spike

proteins to synthesise and then protrude from the surface of endothelial cells. Freely circulating spike proteins can also bind to endothelial cells causing thrombi (blood clots).

Pfizer's novel vaccine excipient ALC-0315, a cationic lipid, is used to aid in the cellular uptake of the LNPs.[72,73] In turn, a second vaccine excipient, ALC-0159, a polyethylene glycol (PEG) lipid conjugate,[72] protectively coats the LNP surface[74] and shields the positive charge to help successfully deliver the genetic cargo into cells. Despite the much-emphasised nimbleness of novel genetic vaccine technology, the safety risk[75] incurred in delivering fragile mRNA molecules in this way is the use of two novel (untested) excipients. For equivalent reasons, Moderna's experimental product contains proprietary ionisable lipid SM-102 and synthetic lipid PEG2000-DMG.[66] As recently as April 2018, the unresolved "problem of toxicity", concerning cationic lipids activating pro-apoptotic and pro-inflammatory cascades was outlined by toxicologists.[76]

Unlike the two mRNA products available in the UK, AstraZeneca's ChAdOx1-S uses a recombinant replication-deficient chimpanzee adenovirus encoding the SARS-CoV-2 spike protein designed to deliberately infect cells with this genetically modified virus[77] and so deliver 50 billion viral particles per dose to instruct cells to produce the spike protein.[78] In study 0841MV38.001,[79] it was shown that the AstraZeneca adenoviral vector distributes to organs including the heart, liver, lymph node, ovary and testes.[80] Megakaryocytes, platelet precursors in bone marrow, may genetically express spike proteins via the adenoviral vector, potentially causing thrombocytopenia.[80]

From a safety perspective, the pathogenesis suggested in the literature calls into question the entire rationale for employing the spike protein as the vaccinal antigen of choice, especially given that it is generated by experimental gene-therapy products produced by manufacturers who have not provided reassuring public pharmacokinetics data. Where also is the data from the manufacturers on the length of the time that the body produces spike proteins? Where is the animal trial data to reassure the public on any aspect of safety? Human trials were, of course, initiated without animal trials to establish efficacy.[81] Very close scrutiny of adverse reactions and autopsies amongst all vaccinees is urgently needed to understand the extent of this spike-protein-induced pathogenesis that is already occurring. Why is this not already happening? Given that Phase III clinical trials are expected to be ongoing until 2023[1,2] in the absence of a control group, routine monitoring of vaccinees for evidence of recent blood clotting events via D-dimer testing should be a priority in the pharmacovigilance strategy of public health bodies, since elevated D-dimer levels are a hallmark of recent microthrombotic events.[82] Urgent questions must immediately be addressed by the MHRA, the UK's medical regulatory who temporarily authorised these experimental products, including Pfizer's product BNT162b2.[83] Why did their temporary use authorisation explicitly require no pharmacokinetics data, despite this being required by Japan's PDMA? The MHRA's own supposed justification for this cites the "immunologic processes" by which vaccines generally work obviating the need for such data.[83] However, this fails to address the critical need to specifically validate the safety profile of the production, biodistribution and pathogenesis of the foreign spike protein generated by the underlying mRNA gene therapy mechanism underpinning any conventional vaccinal anti-spike protein antibody response in BNT162b2.

More generally, why were the two clinical studies in Germany, BNT162-01, and the U.S., C4591001, considered sufficient (for the temporary use authorisation) to assess the product's safety and efficacy for the UK's adult population at large, given that both studies were comprised

of "healthy" test subjects?[83] In BNT162-01, for example, "immunocompromised individuals with known or suspected immunodeficiency" and anyone with a "history of severe adverse reaction associated with a vaccine and/or severe allergic reaction (e.g., anaphylaxis) to any component" were excluded from Phase I, II and III clinical trials.[84] On the basis of such narrowly focussed clinical trials, how can the population at large (with all manner of individual and complex health profiles) be expected, much less compelled, to subject themselves to the risks of taking an experimental product such as BNT162b2? Why also is the drug substance manufacturing process redacted in the FDA's EUA Review Memorandum of BNT162b2?[85] If the government wishes to engender public confidence, in response to Dr Lee Proctor's Freedom of Information request, why has the MHRA invoked both absolute exception Section 41 and qualified exception Section 43 to excuse themselves in not sharing the base nucleotide codes used in the experimental genetic therapies?[86] How can the public be expected to consent to such unprecedented infringements of their civil liberties and voluntarily enlist in genetic experimentation, whilst the chemical structure of the active pharmaceutical ingredient (API)[86] and manufacturing process is kept from public scrutiny? The same questions can be levelled at the MRHA for the two other experimental products they have temporarily approved.

A journal article published as early as May 2020 strongly cautioned against aggressively pursuing a haphazard vaccination program, without paying due attention to immunopathology caused by selecting the SARS-CoV-2 spike protein or nucleocapsid protein as the vaccinal antigen.[87] In vitro experimentation found moderate to strong reactions between anti-spike antibodies and many human tissue antigens, raising the prospect of anti-spike antibodies directly causing tissue damage across the body.[87] The researchers warned that "in the absence of thorough and meticulous safety studies [mass vaccination] may exact a monumental cost on humanity in the form of another epidemic, this time a rising tide of increased autoimmune diseases and the years of suffering that come with them."[88] Since female reproductive tissue both readily expresses the ACE2 gene and shares long linear amino acid sequences with the SARS-CoV-2 spike protein, it itself may be attacked by cross-reactive anti-spike antibodies, a biological mechanism known as molecular mimicry.[89] These autoantibodies may be produced following infection or vaccination and may contribute to autoimmune disease and even infertility.[89]

Numerous other safety concerns over the novel mRNA gene-therapy vaccines have been raised in peer-reviewed journals, including one co-authored by Dr Stephanie Seneff, Senior Research Scientist at MIT.[90] These include concerns that the novel vaccinal excipient PEG could cause anaphylaxis, severe adverse reactions and directly impair vaccine efficacy, given the prevalence of anti-PEG antibodies.[90] One previous study of 200 healthy individuals found a 97.5% prevalence of anti-PEG IgG and IgM antibodies in donor serum.[91] Common prior exposure to PEG may include through the use of cosmetics, non-vaccine pharmaceutical products and processed foods.[91] Even if anti-PEG antibodies do not exist in a given vaccinee, administration of the first mRNA vaccine dose can elicit it, inducing an anti-PEG immune response later recalled in a dual-dose (or booster) vaccination regime.[92] The California Department of Public Health stopped using Moderna's vaccine lot 41L20A due to multiple severe allergic reactions occurring in a single clinic in one day.[93]

A recent study from Greece also concludes that the clinical display of herpes zoster (shingles), via reactivation of the varicella-zoster virus, in a group of immunocompetent individuals represents a "probable" adverse reaction to the Pfizer inoculation.[94] Another significant risk is that vaccinal spike proteins misfold inducing prion-like fatal neurodegenerative diseases.[90] Generally, pathogenic prion proteins that accumulate in the brain cause inflammation and,

ultimately, incurable conditions such as Creutzfeldt–Jakob disease (sCJD), which accounts for 85% of instances of prion disease.[95] Aside from PEG, phospholipids and cholesterol, the LNPs are also composed of ionisable cationic lipids, which both creates an acidic pH and stimulates a pro-inflammatory cytokine response,[96] two predisposing conditions for protein misfolding, further increasing the risk of prion disease.[90] Given that these remain experimental products, LNP cytotoxicity (i.e., cellular toxicity) remains an unresolved safety concern,[97,98] having previously induced injuries to liver and lung organs in rodents.[96]

A yet further risk is vaccinal shedding, whereby vaccinees expel (through bodily fluids, faeces, skin contact and exhalation) something biologically active to people nearby, which could indeed be exosomes containing misfolded spike proteins.[90] Despite the very concept of shedding being strenuously denied by some, Pfizer's own clinical protocol outlines passive exposure events to monitor for during their clinical trials, including:[84]

- *A female family member or healthcare provider reports that she is pregnant after having been exposed to the study intervention by inhalation or skin contact.*
- *A male family member or healthcare provider who has been exposed to the study intervention by inhalation or skin contact then exposes his female partner prior to or around the time of conception.*

Given the second scenario, this suggests that Pfizer accept there is a biologically plausible mechanism for something harmful to be transferred from a vaccinee, through an intermediary host, to a third victim host. Furthermore, as far back in 2015, the FDA itself produced a document to provide industry guidance on the type and extent of shedding data that manufacturers of virus-based gene therapies must collect in their preclinical and clinical trials.[99]

Recent research analysing data from the European Medicines Agency and the Dutch National Register suggests that for every 3 deaths potentially averted by the experimental injections, 2 deaths have already been caused by it.[100] This article was initially published in MDPI's Vaccines journal but was subsequently retracted. In the retraction notice, the journal's editors essentially claimed that since the study authors could not prove a causal relationship between any reported death and vaccination, the article's statistical analysis was invalid.[101] Despite the sheer implausibility of the researchers being able to fulfil such an unreasonable ask, this critique raises logical inconsistencies. Why is this suspicion not equally levelled at deaths that occur in individuals who test positive for SARS-CoV-2 via a RT-PCR test? In the UK, for example, a deceased individual may have any number of comorbidities or even terminal illness and yet the UK government tracks the number of such individuals who returned a positive test result no more than 28 days before dying.[102] Whilst the exact false positive test rate for RT-PCR tests at a given Ct threshold is unknown, recent peer-reviewed Bayesian inference suggests a significant incidence of false-positive results is likely in real world settings.[103] It can thus be argued that, given that like-for-like coincidental deaths not solely caused by either infection or vaccination exist across both data sets, fatal adverse reaction public data is at least as credible as Covid death data, if not far more so. This is because the occurrence of a vaccination prior to death in a vaccinee carries no uncertainty, but it is somewhat uncertain that an individual ever contracted the virus even if they returned a positive test result. Furthermore, it is also a relatively time consuming and complex process to submit a report to a public adverse reporting system such as the MHRA's Yellow Card Scheme, which may dissuade some from submitting a report. There may also be additional influences exaggerating the already known phenomenon of adverse reaction underreporting,[104] because so much of the messaging from the media and public health bodies discounts the possibility that the experimental vaccines are the cause of serious adverse

reactions, apart from in exceptionally rare instances. Such messaging may thus discourage individuals from even contemplating submitting an adverse reaction report. There seems to be far too little time or appetite amongst public health bodies to *rule out* the possibility that these reported vaccine-induced deaths could have been caused by these experimental injections despite this being their responsibility. The onus to prove causality is inexplicably put on academic researchers simply performing association statistical analysis on the public data available to them. Vaccine-induced death reports are thus treated with an overabundance of suspicion. Regardless, given the long-term vaccine-induced pathogenesis already suggested by eminent molecular biologists, where is the evidence that risk-benefit analysis favours vaccination in general, let alone for any one individual?

The 1976 Swine Flu vaccine programme was halted in America after approximately 20% of the population was inoculated because "federal health officials decided that the possibility of an association of GBS [Guillain-Barré syndrome] with the vaccine, however small, necessitated stopping immunization".[105] In an interview on 60 Minutes, Dr David Sencer, head of the CDC during the supposed 1976 epidemic, could not confirm any cases of Swine Flu Influenza A occurred outside of Fort Dix, New Jersey.[106] He was further unable to confirm that the vaccine, X53A, given to most of the public had been tested at all.[106] The consent form given to the public concerned a different vaccine and did not disclose risks of neurological side effects, even though post-vaccination development of neurological complications like GBS was suspected by Dr Michael Hattwick, director of the CDC's surveillance system,[107] who claims he shared his concerns at the time.[106] CDC advertisements even falsely claimed celebrities such as Mary Tyler Moore had taken the vaccine, even though she confirmed she neither took the vaccine nor endorsed the CDC's promotional material.[106] Despite this fiasco, Dr Sencer later served as the Commissioner of Health of the City of New York.[108] Given a history of scandal such as this, why are public health bodies the world over seemingly so immune from the media's scrutiny? GBS is just one of numerous safety concerns in today's experimental mass vaccination campaign. The MHRA does not "rule out" a causal relationship between the AstraZeneca vaccine and GBS[109] following 403 such reports amongst these vaccine recipients recorded via the Yellow Card Scheme reporting system, besides 24 reports of Miller Fisher syndrome, a related nerve disease.[110] PRAC, the European Medicines Agency (EMA) monitoring body, has already recommended adding a safety warning to the AstraZeneca vaccine to alert to the risk of GBS.[111]

At the time of writing, VAERS data indicates that 14,925 people have died following SARS-CoV-2 vaccination in America,[112] whilst 1,645 deaths have been recorded through the Yellow Card Scheme.[110] VAERS represents just one of the adverse event [AE] reporting systems in the US. An independent study on its SARS-CoV-2 data concludes that between the phenomenon of underreporting and reporting lags, the "overall risk signal is high" and suggests that "the vaccines are likely the cause of reported deaths, spontaneous abortions and anaphylactic reactions in addition to cardiovascular, neurological and immunological AEs".[104] The real-world short-term safety data signals are far more concerning than those in 1976, so why has an immediate moratorium on these injections not already been called? Why is the public not being daily informed of the UK's Yellow Card Scheme adverse reaction data in the same manner that Covid-19 cases, hospitalisations, deaths and vaccinations are being keenly reported daily in every media outlet? Given the mechanisms for vaccine-induced pathology, why is the public still so uniformed of the early-warning signals to monitor for post-injection lest they suffer from an acute serious adverse reaction such as a stroke or venous thrombosis?[113] Even microthrombotic complications are known to be a significant contributor to morbidity amongst SARS-CoV-2 sufferers.[82] A peer-reviewed journal study using anatomical and pathological data reports that

91.3% of deceased SARS-CoV-2 patients suffered from microthrombosis and identifies endothelial damage (and the subsequent immune response) as a key driver in severe SARS-CoV-2 disease.[114]

An in vitro study targeting Primary B cells, K562 cells (derived from monocytes), and Raji cells (cultivated B lymphocytes) found that SARS-CoV-2 viral infection was enhanced in suboptimal antibody neutralisation concentrations,[115] as may be expected in vaccinal antibody levels in the months following injection – especially amongst the immunocompromised. This is significant because the mechanism of antibody-mediated productive infection (trophism) of such phagocytic immune cells is a known contributor to vaccine-associated enhanced disease (VAED).[116] Whilst it is a preprint, this study has already been cited multiple times in peer-reviewed scientific journals. A key finding is that anti-spike antibodies have the potential to first bind to the SARS-CoV-2 virus and yet *enhance* the subsequent membrane fusion reaction between the virus membrane (via the S2 subunit) and the host-immune-cell membrane, increasing the likelihood of viral entry into these immune cells, and so promoting cellular infection and ensuing viral replication.[115] Specifically, the binding of the Fc domain of anti-spike IgG antibodies with the FcγRII antibody receptors of the Raji cells was implicated in this antibody dependent enhancement (ADE), a mechanism similar to that previously observed for previous coronaviruses SARS-CoV and MERS-CoV, Zika and dengue virus.[115] Even though ADE has not yet been unequivocally demonstrated in vivo with respect to SARS-CoV-2, it is still entirely impossible to project into the future of this pandemic and conclude that it will never be identified in individuals with low antibody neutralisation levels, or against a new viral strain yet to dominate, or in a cross-reactive vaccinal antibody response against a CoV causing the common cold, the latter of which will probably take "several years" to investigate.[117]

Importantly, SARS-CoV-2 vaccinal antibodies not only target the spike protein's S1 RBD, but also its N-terminal domain (NTD) and S2 subunit.[118] Very recent molecular modelling simulations suggest that ADE *in vivo* may occur with yet-to-emerge viral strains (and even with the Delta variant) due to antibodies stabilising the binding of the virus' spike protein to host cell membrane at its NTD. It is hypothesised that this NTD-directed facilitation will thus promote conformational change in the spike protein and so enhance cellular viral entry (and ensuing replication) via the RBD post-conformation.[119] ADE is not easy to predict, because it need not only be mediated by the choice of the spike protein as the vaccinal antigen, but it may be due to a given vaccine adjuvant or due to the age of a subset of recipients.[120] Given all of this uncertainty, why are our political leaders so determined to *seroconvert* the entire population? Are all our political leaders so supremely (or, divinely) confident of the long-term prognosis for all vaccinees fighting all manner of future CoV infections, in viral strains yet to even emerge, in the face of all this research, even with all these unanswered questions?

Vaccine Theory and Efficacy

Previous studies on coronavirus vaccines provide clear warnings about the potential risk of enhanced respiratory disease (ERD) and ADE. In ADE, targeted cells are infected at a higher rate, or disease worsens due to enhanced immune activation. ERD can complement ADE or can be associated with biological mechanisms independent of antibodies, including "cell death, cytokine release and/or local immune cell activation."[121] In 2012, four candidate human vaccines against SARS-CoV were trialled in mice experiments.[122] Whilst the vaccines initially induced antibody responses to protect against infection, upon exposure to the wild virus, ERD immunopathology was observed.[122] The conclusion of the researchers was stark: "Caution in proceeding to application of a SARS-CoV vaccine in humans is indicated."[122] As recently as 2019, the SARS-CoV anti-spike IgG antibody response of vaccinated Chinese rhesus macaques subsequently challenged with the virus did reduce viral replication, but ADE induced severe acute lung injury (ALI) by "skewing" the macrophage immune response.[123] A literature review published in 2021 in *Nature Reviews* Microbiology concluded: "The adverse reaction rate of a COVID-19 vaccine should be kept extremely low if it is distributed globally."[124] A necessary three-fold criteria for developing a safe and effective coronavirus vaccine were well summarised in the 2013 PhD dissertation of virologist Meagen Deming (née Bolles).[125] In the below table, the current mass vaccination campaign is assessed against these three criteria.

Deming's PhD Vaccine Criteria	**First-generation genetic vaccines against SARS-CoV-2**
Cross-protectivity against heterologous viral variants	All first-generation genetic vaccines narrowly target the spike protein of the initial Wuhan viral strain. Decreased effectiveness at reducing infection against current dominating Delta strain.[6] Forecasting from molecular and genomic epidemiologists suggests the virus is likely to evolve towards full immune escape.[126–128] Even revaccination may fail to update the antibody response due to original antigenic sin, as has been observed previously with flaviviruses.[129–131]
Robust immune responses in immunocompromised elderly recipients as these are at highest risk of suffering morbidity and mortality from the virus	Even amongst the general population, vaccinal antibody levels are waning and these antibodies are less neutralising against the Delta variant.[132,133] Delta breakthrough infections are significantly more common than breakthrough infections observed in the clinical trials.[6,16,17] Case studies and government data suggests that elderly immunocompromised double-dose recipients continue to be at risk of contracting the virus, developing severe disease and dying.[134,135] Viral loads in infected vaccinees are equivalent to those in infected unvaccinated individuals.[8–10,136]
Avoidance of adverse events	Spike protein produced genetically freely circulates[52] and acts pathologically against the body independently,[137] causing micro, severe and fatal thrombotic events and other complications. Researchers warn of risk of significant increase in serious pulmonary and cardiovascular disease.[57]

A safe and effective SARS-CoV-2 vaccine must thus not only avoid adverse events, but also induce a durable immune response amongst those most likely to succumb to the disease that is *cross-protective* against all viral variants that emerge under pandemic conditions.[138] *Cross-reactive* anti-spike antibodies that bind to a viral protein, but fail to neutralise the virus and prevent cellular infection, are not protective. Such antibodies could even enhance the cellular infection in an ADE scenario.

Given the specific anti-spike antibody vaccinal immune response; indefinitely, intrusive global health surveillance measures to detect emerging and newly proliferating immune escape viral variants are required.[139] Should such strains begin to dominate, strict infection control measures and repeated inoculations could be attempted to retrain vaccinees' immune responses against epitopes characterising the circulating immune-escape viral strains. Despite the risk of further vaccine-induced side effects, this may seem to be an acceptable compromise, but does it even represent a viable strategy? Is the proliferation and dominance of immune-escape variants even likely and, if it does occur, should it concern us? Is revaccination with genetically updated spike proteins certain to be efficacious? These are the urgent questions that must now be addressed.

It is immediately apparent that the immune response elicited by natural infection is broader than that induced by use of monoantigenic genetic vaccines. Many studies already suggest that natural infection confers protective immunity against reinfection.[140] A recent Israeli preprint has found that naturally infected individuals are up to 13 times less likely to be reinfected with the Delta variant than vaccinees not previously infected and up to 27 times less likely to develop symptomatic disease.[141] In another study on unvaccinated healthcare workers, convalescent plasma tested 10 months after infection suggests sustained protection against reinfection, with an estimated rate of just 7% IgG decay per month since infection.[142] Similarly, an Austrian study found stable IgA antibody levels and 76% of initial IgG levels retained in convalescent plasma eight months following the initial measurements.[143] Since the experimental gene therapies encode for and generate the original (Wuhan) spike protein, only antigenic epitopes on this spike protein can possibly trigger a T or B cell mediated immune response. By contrast, natural infection exposes the host to the entire virion, including to non-structural proteins and the four major structural proteins: spike, nucleocapsid, membrane and envelope. Naturally infected individuals are known to mount robust antibody responses to both the spike protein and the nucleocapsid protein.[144] Key areas where natural and acquired immunity must be thus contrasted are memory B cells, T helper cells (CD4+ T-cells) and cytotoxic T cells (CD8+ T-cells).

A study published in November 2020 identified the breadth of cytotoxic T cell immunity acquired through natural infection.[145] Whilst neutralising antibodies may wane following natural infection, memory cytotoxic T cells can be far more durable.[145] Human leukocyte antigen (HLA) epitopes present specific viral peptide sequences that are recognised by virus-specific memory CD8+ T-cells.[145] In the case of SARS-CoV, these CD8+ T-cells can persist for 6-11 years and are more durable than memory B cells and antiviral antibodies.[145] The HLA epitopes in this same study were found to *not* be liable to mutation and only 3 of 29 shared epitopes, observed across patients in the study, were located in the spike protein.[145] Most of the others were found in the ORF1ab or the nucleocapsid protein, implying a far broader natural ability to kill infected cells.[145] Thus, naturally acquired memory CD8+ T-cells are likely to offer broad and durable cytotoxic T cell immunity, which is a very important line of defence for limiting disease caused by a viral infection. By contrast, vaccine-elicited cytotoxic T-cells can only possibly recognise epitopes in the spike protein.

T helper cells can suppress viral infection, induce CD8+ T-cell responses and activate B-cells to produce antibodies. Naturally acquired memory CD4+ T-cells also *broadly* target several SARS-CoV-2 proteins equally and have already been shown to persist in an 8-month longitudinal study.[146] Again, vaccine-elicited T helper cells can only target the mutable spike protein. A very recently published immunology paper suggests that, based on the breadth of T cell responses alone, polyantigenic vaccines that do not just target the spike protein but other proteins including the ORF1ab non-structural protein would likely better counter emerging viral variants.[147] Required for viral genome transcription, ORF1-encoded proteins are known to be produced first in CoV-infected cells. It has therefore even been theorised that ORF1-specific T cell activation could intervene to break down (lyse) infected cells prior to the formation of mature SARS-CoV-2 virions, potentially further suggesting their importance in rapid cellular immunity against SARS-CoV-2.[148]

Recent experimental data on vaccinees receiving Pfizer's product BNT162b2 found that the injections induce complex functional changes in their innate immune responses.[149] Toll-like receptors (TLRs) are pattern recognition receptors (PRRs), which are critical in identifying harmful patterns in pathogens before inducing immune inflammatory defences via signalling pathways.[150] TLR4 recognises bacterial lipids and TLR7/8 recognises viral RNA.[150] The experiment showed that the TLR7/8 agonist (activator) R848 led to a statistically significant decreased immune response expressed by TNF-α inflammatory cytokines and a decreased TNF-α response to the TLR3 agonist poly I:C.[149] It is possible that innate immune changes such as this could "diminish antiviral responses."[149] Conversely, anti-inflammatory cytokine responses to fungal pathogens increased,[149] reflecting a further change in the innate immune response. For subpopulations with broad, robust innate immune systems such as immunocompetent children, this alone suggests that vaccination alone could introduce risks to their healthy innate immune systems fighting pathogens even unrelated to coronaviruses. Children's strong antiviral innate immunity in the upper airways is generally able to efficiently clear SARS-CoV-2 infections.[151] The mantra of mass vaccination, enforced through coercion and social engineering, even compels previously infected individuals to be subject to the experimental injections with potentially harmful consequences.

Epidemiology

A landmark 2004 paper outlying a "phylodynamic" framework to describe the evolution of RNA viruses under epidemic conditions theorises that viral adaptation occurs at the highest rate under intense immune-selection pressure and high infectious pressure.[20] Today, genetic vaccines are narrowly targeting the SARS-CoV-2 spike protein on a background of high infectious pressure[10] from the virulent and dominant Delta strain. As early as October 2020, researchers demonstrated that convalescent plasma taken from recovered patients containing "unknown" levels of neutralising antibodies against SARS-CoV-2, presumed to mostly be at suboptimal concentrations, soon led to antibody-resistant mutations in vitro.[152] At this stage of the mass vaccination campaign, we can identify at least three groups contributing to such immune-selection pressure: individuals who so far have received only one injection, those with waning antibody levels in the months following their last injection, and immunocompromised individuals receiving blood plasma from recovered patients. In the absence of strict infection control measures, recent statistical modelling suggests that the present times thus provide optimal conditions for the establishment of immune-escape variants.[153] Indeed, data from California already suggests that fully vaccinated individuals are significantly more likely than unvaccinated (77.6% vs. 47.7%) to be infected with antibody-resistant SARS-CoV-2 variants.[154] A recently published genomic sequencing study from Israel compared unvaccinated subjects with those that received BNT162b2. It found a statistically significantly higher rate of vaccinees contracted VoC B.1.1.7 (Alpha) following their first dose and a statistically significantly higher rate contracted VoC B.1351 (Beta) following their second dose, as compared with unvaccinated infected subjects.[155] As per quasispecies theory, vaccinees exert positive selection pressure to promote the dominance of those viral variants that tend to evade antibody neutralisation. Virologist Prof Luc Montagnier, co-discoverer of HIV and 2008 Nobel Prize Winner in Medicine, stated in a video interview translated and published by the RAIR Foundation US, "It's an enormous mistake, isn't it? A scientific error as well as a medical error. It is an unacceptable mistake."[156] He also said, "The history books will show that because it is the vaccination that is creating the variants" and, "It is clear that the new variants are created by antibody-mediated selection due to the vaccination."[156] On the fundamental question of vaccinating during a pandemic, "Many epidemiologists know it and are 'silent' about the problem known as 'antibody-dependent enhancement.'"[156]

Furthermore, the virus can already cross the species barrier, infecting mink[157] and can likely infect many other species including rabbits.[158] There are thus many mammalian species kept in high-density housing, which would promote high infectious pressure in the event of an outbreak, potentially breeding new variants that can again reinfect humans. Any global mass vaccination strategy attempting to out-vaccinate the virus amongst humans must also account for the many susceptible animal hosts and their related interactions with humans. Vaccinating animal hosts with similar non-sterilising vaccines[159] would likely exert additional positive selection pressure.

Aside from the emerging threat of immune escape, another worrying phenomenon is the increasing infectiousness of circulating SARS-CoV-2 viral strains. Non-sterilising vaccines that facilitate viral transmission risk increasing viral virulence,[160] as has been observed previously in Marek's disease in chickens.[161] Recent data from Vietnam shows that median and peak viral loads were respectively 215 and 251 times higher for breakthrough Delta infections amongst vaccinated healthcare workers compared to infections of SARS-CoV-2 strains circulating in March and April 2020.[8] The study also found that viral RNA loads derived from mean Ct (cycle

threshold) values using quantitative RT-PCR testing were not statistically significant between unvaccinated cases and fully vaccinated breakthrough cases.[8]

Anti-spike neutralising antibodies protect cells from infection by binding to the spike protein's RBD and NTD domain and S2 subunit.[118] For antibody-to-spike-protein binding and spike-protein-to-ACE2-receptor binding, the RBD is the common denominator. Mutations that reduce the affinity of anti-spike antibodies for the RBD, without reducing the affinity of the spike protein to the ACE2 receptor will thus increase viral infectivity. Two mutations E484 and S494p for amino acid residues at RBD positions 484 and 494 respectively have already been identified as having this property, with S494P prevalent in 0.81% of all sequenced genomes as of March 2021.[162] In August 2021, Portuguese epidemiologists, under the editorship of the distinguished academic Prof Michael S Diamond, suggested that the prevalence of these mutations will likely increase as a result of anti-spike antibodies acquiring in seroconverted individuals (i.e., principally as a result of the spike-protein genetic vaccines), and therefore should be closely monitored.[162] Another recent epidemiology preprint, with contributions from 23 scientists from across the world, warns that through examination of SARS-CoV-2 genomes, they now find evidence of "major changes in the selective forces" on genes including the spike protein gene.[128] They further warn that the N501Y lineage, featuring the N501Y spike mutation that enhances viral infectivity and transmissibility by strengthening RBD-to-ACE2 binding affinity[163] and present in the Alpha, Beta and Gamma variants of concern (VoC) but not Delta,[164] is evolving a survival strategy to evade vaccine-induced hostile conditions.[128] In suggesting in vitro experimentation to confirm their concerns,[163] they also identify the threat of rapid genetic recombination[128] where two distinct viral strains combine in a single host, sometimes with dramatic effect.[26] They posit that this could lead to the emergence of new strains that could be a "considerably bigger problem for us than any we currently know" due to the potential for any combination of increased infectivity, transmissibility and anti-spike immune evasion.[128] Indeed, the challenging combination of higher infectiousness, increased resistance to neutralising antibodies and increased susceptibility to infection-enhancing antibodies has already characterised the rapid infection spread of the Lambda variant in South America, even in nations such as Chile with a high background vaccination rate – approximately 60% of its population has received at least one dose.[165]

A late August 2021 preprint from Japan warns that the Delta variant can readily acquire full resistance to vaccinal anti-NTD anti-spike antibodies acquired against the original viral strain encoded in the first-generation genetic vaccines, given their experimental results on mice.[126] The researchers deliberately introduced four common amino acid substitutions K417N, N439K, E484K and N501Y in the RBD into the Delta variant to artificially create a Delta 4+ pseudovirus, and found that it completely evaded anti-NTD anti-spike neutralising antibodies, but maintained the epitopes recognised by enhancing anti-NTD anti-spike antibodies, which act to inhibit anti-RBD anti-spike neutralising antibodies.[126] Their results show how a relatively small number of mutations can induce a subtle change (i.e. in the delicate balance of neutralising vs. enhancing anti-NTD anti-spike antibodies), which in turn induces a dramatic effect on infectivity, with potentially severe consequences. Besides the kind of RBD mutations introduced in the Delta 4+ pseudovirus, another peer-reviewed publication has identified amino acid deletions in the S1 NTD, at the recurrent deletion regions (RDRs) in the domain targeted by anti-NTD anti-spike antibodies, thus obliterating the NTD epitopes the antibodies were elicited to target, blunting the action of anti-spike neutralising antibodies generated through vaccination or otherwise, and so helping the virus to evade the immune system.[166] The article's authors conclude that it may be "critical but overlooked" to diversify therapeutic targets beyond just the

mutable spike protein.[166] Given that recent statistical analysis suggests that current VOCs, with their many mutations, appear to have emerged due to episodic increases in the evolutionary rate 4-fold higher than the background rate, it is important to understand the conditions under which challenging variants emerge, proliferate and dominate.[167]

Every day, an ever-increasing proportion of the population is newly seroconverted through vaccination and so gradually acquire highly specific neutralising anti-spike antibodies. Simultaneously, neutralising antibodies wane in other parts of the population. In some immunocompromised individuals, vaccinal antibodies were never robustly elicited. Irrespective of the specific event leading to its emergence, how long will it be until all this immune-selection pressure exerted by vaccinees with immature anti-spike antibody responses leads to the widespread proliferation and dominance of immune-escape variants significantly more devastating than all current VoCs?

Original antigenic sin (OAS) describes a phenomenon where cross-reactive antibodies from an initial infection are recalled to target an epitope in a secondary related but distinct pathogen. A recent preprint study using patient blood samples identifies antibodies initially produced against an antigen on the neuraminidase protein of the widespread H3N2 Influenza A virus of 2014 as the likely source of immunological memory inducing anti-Ep9 antibodies elicited to fight SARS-CoV-2.[168] There is a known "strong association" between such antibodies that bind to the Ep9 epitope region of the SARS-CoV-2 nucleocapsid protein and severe disease outcome related to cytokine hyperactivity.[168] A second example of OAS has been observed whereby patients infected with SARS-CoV-2, having previously been infected with human coronaviruses HCoV-HKU1 or HCoV-OC43, strongly recall cross-reactive non-protective HCoV antibodies at the expense of IgG and IgM anti-spike and anti-nucleocapsid antibodies targeting SARS-CoV-2.[169] Memory B cell activation thus overwhelms and hinders newly-activated naïve B cells.[131] In this way, immunological memory can impair the humoral immune response.

If immune-escape mutations in the SARS-CoV-2 spike protein RBD or NTD domain characterise a new dominant strain, first generation vaccine-induced antibodies will no longer all adequately neutralise the virus. In an OAS scenario, such an attempt to update the immune response by revaccinating with the genetically up-to-date spike protein may thus recall and elicit the production of non-neutralising first-generation anti-spike antibodies at a significantly higher rate than second-generation anti-spike antibodies. Protective, neutralising second-generation anti-spike antibodies can thus be overwhelmed due to OAS recalling first-generation cross-reactive non-neutralising antibodies targeting conserved spike protein epitopes common to both generations of spike proteins.[130] If this risk is realised, OAS could exert a catastrophic impact on the ability of (even healthy) vaccinees to fight SARS-CoV-2 infections. The danger is exaggerated given the increasing contagiousness of infectious variants, also making it difficult to prevent many vaccinees from becoming infected. The propensity for RNA viruses such as SARS-CoV-2 to adapt their antigenic surface to evade vaccine-induced antibody-antigen neutralisation and the associated risk of OAS was already a known problem for implementing a universal vaccine strategy prior to the emergence of SARS-CoV-2.[129] Other enveloped RNA viruses that have previously elicited OAS during revaccination include flaviviruses tick-borne encephalitis, yellow fever and dengue fever, as well as the non-enveloped DNA virus group human papillomavirus (HPV).[130]

Prophylaxis and Early Intervention Treatment

Beyond the laser focus concentrated on the experimental mass vaccination campaign, why is mention of antiviral medication and repurposed drugs[170] as both prophylaxis and early intervention treatment, so distinctly lacking in public health messaging? Where is the advice for infected individuals to immediately intervene therapeutically upon the first hint of infection with safe and effective over-the-counter antiviral medication, vitamin supplementation or monoclonal antibodies,[171] especially for those most at risk? Why do we continue to passively allow the virus to replicate in infected (and even vulnerable) hosts and cause disease without an intense, conventional antiviral challenge? Why are those infected encouraged to wait at home until they present with symptoms requiring hospitalisation, such as when the virus has taken such a foothold that they experience shortness of breath, sometimes having blood-oxygen levels so low they are hypoxic?

By 2010, it was known that increased intracellular Zn^{2+} concentrations delivered via a zinc-ionophore significantly impairs RNA viral replication in vitro for viruses including poliovirus, influenza and coronavirus.[172] As early as April 2020, Derwand and Scholz suggested combining chloroquine (or hydroxychloroquine) with zinc supplementation to challenge SARS-CoV-2 infections, citing the advantages of such a treatment as availability, affordability, efficacy and safety.[173] These German medical experts subsequently collaborated with Dr Vladimir Zelenko to document the success he observed in a New York community between March and May 2020.[174] Relative to a control group, a statistically significant decline in hospitalisations (84%) was observed in the treatment group subject to a triple drug protocol consisting of zinc, hydroxychloroquine and the antibiotic azithromycin.[174] By July 2020, independently, three other expert German immunologists jointly concluded that prophylaxis treatment with zinc for vulnerable cohorts should begin immediately, even as formal clinical trials on zinc protocols against SARS-CoV-2 were ongoing.[175] Besides inhibiting RNA viral replication, they provided other examples of the benefits of zinc supplementation, including reducing the risk of hyper-inflammation, preserving the epithelium (which can be damaged by the spike protein), anti-oxidative effects and the supporting of antiviral immunity.[175]

In December 2020, consultant cardiologist and professor of medicine Dr Peter McCullough and others published on early drug treatment protocols, collating the many actual protocols using chloroquine/hydroxychloroquine worldwide – across Central and South America, Africa, Asia, and Europe.[176] In the following month, Dr McCullough collaborated with others to again publish a paper on the benefits of early intervention outpatient treatment on the outcome of disease caused by SARS-CoV-2.[177] Other peer-reviewed research has made very similar recommendations, sometimes with a specific pathophysiological emphasis, such as: SARS-CoV-2 should be treated early to prevent hypersensitive immune dysregulation causing disease in vulnerable patients.[178] Used primarily against mental health conditions such as OCD, ADHD and depression, the drug fluvoxamine acts as a selective serotonin reuptake inhibitor (SSRI). However, it also decreases cytokine production, including IL-6,[179] reducing the risk of a dangerous overabundance of pro-inflammatory cytokines generated during a cytokine storm.[180] The IL-6-STAT3 signalling pathway has been implicated in the cause of cytokine storms leading to acute respiratory distress syndrome (ARDS), a severe form of SARS-CoV-2 disease.[181] Given its known suppressive mechanism on IL-6, fluvoxamine represents a theoretically credible early intervention treatment. Two small-scale studies on the use of fluvoxamine in peer-reviewed journals show very promising results. In the first study, a double-blind randomised clinical trial,

none of 80 treated outpatients experienced clinical deterioration defined as shortness of breath (requiring hospitalisation or otherwise), pneumonia, oxygen saturation below 92%, or need for supplemental oxygen to maintain oxygen saturation above 92%.[182] In the second study, none of 65 treated outpatients required hospitalisation.[183] Ivermectin[184] represents a third very important option for early intervention.[185] Research published in the Journal of Antibiotics summarises four main roles by which this anti-parasitic medication acts to inhibit SARS-CoV-2 viral infection and the development of its ensuing disease.[186] Among its many mechanisms of actions, it prevents the virus from entering cells by docking to both the spike protein and the ACE2 receptor,[187] it has antiviral ability to inhibit RNA viral replication by binding with RNA polymerase, it inhibits the production of pro-inflammatory cytokines, and it increases mitochondrial ATP, improving cardiac function and preventing heart failure.[186] Excitingly, mass ivermectin treatment in Peru led to an average 74% reduction in excess of deaths in the ten states that most extensively deployed it.[188] Taken as prophylaxis treatment amongst healthcare workers, a weekly dose of ivermectin led to an ARR of 1.8% and RRR of 74%.[189] Unlike the first-generation experimental genetic vaccines, the action of these drugs is independent of highly specific epitopes on the spike protein.

An antiviral diet that supports the natural immune system could also help to clear a SARS-CoV-2 infection prior to the manifestation of serious disease. Nigella sativa, garlic, cinnamon, liquorice root, black pepper, moringa oleifera, mushroom, probiotic yoghurt, honey[190] and spirulina have all been proven to be effective antiviral foods against previous RNA viruses.[191] Their mechanisms of antiviral actions include hindering viral attachment to host cells, increasing T helper cells and cytotoxic T cells, and inhibiting RNA-polymerase-II, the enzyme that initiates RNA transcription when an RNA virus synthesises its viral proteins in an infected host during the viral replication phase.[191] Targeted dietary supplementation including zinc,[192] vitamin D,[193] vitamin C,[194] N-acetylcysteine (NAC),[195] circumin, cinnamaldehyde, allicin, piperine, selenium, propolis, probiotics, lactoferrin, quercetin[194] and glutathione[58] may all help to fight an infection.[196] Given that the experimental SARS-CoV-2 gene-therapy vaccines are in clinical trials until 2023 and do not have long-term safety profiles, why are safe antiviral foods and vitamins not clearly being suggested as potentially helpful prophylaxis therapies for the unvaccinated population by public health bodies? Given further that a study of 48,440 adult patients conducted at Kaiser Permanente Southern California (KPSC) determined a "strong association" between "meeting physical activity guidelines" and reduced risk of suffering a severe SARS-CoV-2 clinical outcome amongst infected adults,[197] why do public health bodies refrain from clearly recommending regular exercise as a safe and potentially important prophylaxis therapy that may help to guard against developing severe SARS-CoV-2 disease?

Conclusion

According to Dr Francis Boyle, professor of international law at the University of Illinois College of Law and responsible for drafting the Biological Weapons Anti-Terrorism Act of 1989 (BWATA), SARS-CoV-2 is an offensive Biological Warfare Weapon.[198] In October 2014, the Obama White House instigated a U.S. government funding pause on gain-of-function research experimentation into viruses to enhance the pathogenicity and/or transmissibility of respiratory viruses influenza, MERS and SARS.[199,200] However, in December 2017, the funding pause was removed as the National Institutes of Health (NIH) instituted the U.S. Department of Health & Human Services (HHS) framework 3PCO to guide funding decisions on any potential pandemic pathogens (PPPs), which are pathogens that are intentionally manipulated to be more transmissible and/or virulent (infectious).[201,202] An August 2021 House Foreign Affairs Committee report provides evidence that gain-of-function research funded by the American and Chinese governments at the Wuhan Institute of Virology focused on increasing the infectivity of the coronavirus spike protein, i.e. by increasing RBD-to-ACE2 binding affinity.[203] Three out of the seven coronaviruses known to infect humans, SARS-CoV, MERS-CoV and SARS-CoV-2, have newly emerged between 2002 and 2019.[11] Could there be a relationship between Chinese and US government-funded gain-of-function research and the sudden emergence of increasingly transmissible and/or pathogenic novel coronaviruses?

Aside from the question of how and why this virus emerged, why was humanity locked down in 2020 to avoid a virus only to emerge to a new pathogen in 2021? Vaccinees now create the viral protein that alone is known to damage the human body. In the absence of long-term safety data and the wholesale suppression of both short-term safety signals from public adverse reaction reporting systems and the warnings from toxicologists and molecular biologists, the prognosis for humanity at large is unknown but appears increasingly bleak unless a dramatic change is instigated.

What could possibly explain the aggressive campaigns of governments and public health bodies across the world to coerce their populations to enlist in medical and genetic experimentation? Why are they so willing to stake public confidence in vaccines on such novel technology? Why is there still such a rush to simultaneously inject the world's populations with these non-sterilising gene-therapy vaccines which neither stop infection nor block transmission? Why are these experimental interventions still heralded as our salvation when they cannot confer cross-protective immunity that protects vaccinees from all future viral variants? Why are safe and effective early intervention treatments still suppressed? Prior to the mass vaccination campaign, the field of epidemiology already informed us of all the pitfalls we now find ourselves in. The scale and complexity of collective human intervention in this pandemic is entirely unprecedented in human history and now threatens our world at large. Whatever the future holds, there can be no doubt that continued reliance on genetic vaccines that solely target the spike protein is untenable. Despite being the premise of the mass experiment, herd immunity has not been acquired and cannot be acquired with the current strategy. The vaccines simply blunt SARS-CoV-2 infections, preventing serious disease and death in some infections, whilst inflicting serious and fatal vaccine adverse reactions in others. The vicious circle of declining anti-spike neutralising antibodies, revaccination and transmission, whilst the pool of vaccinees is ever widened as increasingly younger children are enlisted, can only lead to increasingly immune-escape viral variants to proliferate and dominate. Far from being a triumph of science,

intellectualism and morality, this experiment has turned into something coercive, dangerous and even lethal.

Dr Geert Vanden Bossche, an independent vaccine expert and a former academic at universities in Belgium and Germany, who has since served in various R&D and senior program roles at GSK Biologicals, Novartis Vaccines, Solvay Biologicals, Bill & Melinda Gates Foundation and GAVI, has been an outspoken critic of the mass vaccination campaign. Dr Vanden Bossche has consistently warned the world of the devastating impact of mass vaccination with non-sterilising vaccines on a background of high infectious pressure. Had his warnings been heeded months ago, humanity might not be facing the crisis it finds itself wrestling with today. On 6 March 2021, Dr Vanden Bossche published an open letter on his website to appeal to the World Health Organization (WHO) to immediately open the channels for scientific debate and declare a public health emergency of international concern, given the paradigm of mass vaccination ever pressurising the spike protein towards full immune escape.[204] On 10 March 2021, Dr Vanden Bossche gave a keynote speech at the Vaccine Summit Ohio 2021 on why the SARS-CoV-2 vaccines must not be used in a mass vaccination campaign during the pandemic.[205] On the following day, he directly addressed the WHO via a video, urging an open scientific hearing on the dangers of implementing strict infection control measures and mass vaccination with non-sterilising prophylaxis vaccines on a background of high infectious pressure. Dr Vanden Bossche has not wavered from his thesis on the folly of the current strategy. Regrettably, his thesis is increasingly being vindicated through the research of molecular and genomic epidemiologists. Why was an independent expert as qualified, insightful and impartial as Dr Vanden Bossche not given a platform to address global health leaders back in March 2021? What scientific rationale could possibly justify freezing out eminently credible experts from expressing their concerns, with the stakes so high, especially when their theories are fully supported by sound scientific arguments?

In closing this report, it is appropriate to tell the story of Maddie de Garay. Along with her two brothers, this previously healthy 12-year-old girl received her second Pfizer dose on 20 January 2021 as part of the clinical trials for adolescents. According to Pfizer's press release on 31 March 2021 announcing "positive topline results" for the Phase III clinical trial in 2,260 adolescents, the vaccine achieved "100% efficacy" and "was well tolerated".[206] According to their website, "Pfizer is committed to improving the health and well-being of children through thoughtfully designed clinical trials."[207] According to the CDC, no serious adverse event reported in the adolescent trials for Pfizer's product BNT162b2 was judged by the FDA to be related to the experimental injections.[208] According to the clinical trial report published in The New England Journal of Medicine, "Few participants in any cohort (≤0.4% through 1 month after dose 2) had serious adverse events, and none were considered by the investigators to have been vaccine-related."[209] However, on 28 June 2021, Senator Ron Johnson held a press conference featuring Maddie de Garay and her mother Stephanie.[210] In the video, Maddie is seen sitting in a wheelchair with a nasogastric tube taped on her face. Stephanie's account of Maddie's sudden onset of severe symptoms immediately following her second injection is fully reproduced on the next page. Between the FDA, the CDC, Pfizer, the New England Journal of Medicine, the media, the U.S. government and global health organisations, something is deeply wrong. Why even test a product if a life-changing serious adverse reaction is simply covered up in the clinical trials?

"On January 20th, Maddie received her second dose of the Pfizer Covid vaccine as a participant in the clinical trial for 12 to 15 year olds.

All three of our kids volunteered and we're excited to participate in the trial as a way to help us all return to normal life. My husband works in the medical field and I have a degree in electrical engineering. We are pro-vaccine and pro-science, which is why we agreed to let Maddie and her two older brothers volunteer for the trial.

Before Maddie got her final dose of the vaccine, she was a healthy 12-year-old who got straight A's and had lots of friends. She had a life. She was energetic. She was not like this, although she does still have lots of friends. Upon receiving the second shot, Maddie immediately felt pain at the injection site and over the next 24-hours she developed severe abdominal and chest pain, and the way she described the chest pain, and I quote, 'It feels like my heart is being ripped out through my neck.' She had painful electrical shocks down her neck and spine that forced her to walk hunched over. She had extreme pain in her fingers and toes; it actually made them turn white and they were cold whenever you touched them. She had edema, so my husband immediately took her to the ER as instructed by the vaccine trial nurse administrator, which is what we were instructed to do. Her blood was taken for a renal profile and tested. She was checked for appendicitis, which she did not have, and given an IV with some medicine, then sent home. However, in the discharge papers from the children's hospital ER that she went to, the diagnosis stated 'Adverse effect of vaccine initial encounter.' This would be the only time that that was written in her medical charts, but it's in there.

Over the next two and a half months her abdominal muscle and nerve pain became unbearable. She developed additional symptoms that included gastroparesis, nausea and vomiting, erratic blood pressure, and heart rate, memory loss; she mixes up words, brain fog, headaches, dizziness, fainting – she fell and hit her head – and then seizures. She had verbal… she developed verbal and motor tics. She had loss of feeling from the waist down and muscle weakness, drastic changes in her vision, urinary retention, and loss of bladder control, severely irregular and heavy menstrual cycles and eventually she had to have an NG tube put in to get nutrition. All of these symptoms are still here today, some days are worse than others. Our greatest challenge came when her doctors began to consider an alternative diagnosis… well she really didn't have one before so it was the first one. So, like everybody else she had lots of tests, but not nearly as many tests as everybody else, and she's a child. Why didn't they do all those tests on her? Sorry… So because they couldn't figure it out one physician labeled her as having 'functional neurologic disorder,' saying it was due to anxiety. This concerned us, and we didn't agree with it because she doesn't have anxiety. Look at her. I mean what 13-year-old can sit here calmly if they have anxiety or mental issues? At one point they even tried to admit her to a mental hospital. So we did seek additional medical opinions some of which came from this group.

In June, we connected her neurologist with another doctor that's doing research on adverse reactions like Maddie's. She was finally provided, but they finally gave her an MRI of her brain, an MRV and a bunch of additional blood tests. It took five months to get that done. Over the past five months Maddie has been into the ER nine times and has been hospitalized three times for a total of two months in the hospital.

What I want to ask, Maddie volunteered for the Pfizer trial, why aren't they researching her to figure out why this happened so other people don't have to go through this? Instead they're just saying it's mental. If anybody's mental, it's me. So today our journey as parents to help our daughter Maddie continues. All we want is for Maddie to be seen heard and believed, because she has not been and we want her to get the care she desperately needs so that she can go back to normal. Why is she not back to normal? She was totally fine before this. She did the right thing trying to help everybody else and they're not helping her."[211]

References

1. ModernaTX Inc. A Study to Evaluate Efficacy, Safety, and Immunogenicity of mRNA-1273 Vaccine in Adults Aged 18 Years and Older to Prevent COVID-19. ClinicalTrials.gov. Published June 10, 2021. Accessed September 6, 2021. https://clinicaltrials.gov/ct2/show/NCT04470427?term=NCT04470427&draw=2&rank=1
2. BioNTech SE. Study to Describe the Safety, Tolerability, Immunogenicity, and Efficacy of RNA Vaccine Candidates Against COVID-19 in Healthy Individuals. ClinicalTrials.gov. Published August 26, 2021. Accessed September 6, 2021. https://clinicaltrials.gov/ct2/show/NCT04368728?term=NCT04368728&draw=2&rank=1
3. Deoni SC, Beauchemin J, Volpe A, Dâ Sa V, RESONANCE Consortium. Impact of the COVID-19 Pandemic on Early Child Cognitive Development: Initial Findings in a Longitudinal Observational Study of Child Health. *medRxiv: the preprint server for health sciences*. Published online August 11, 2021. doi:10.1101/2021.08.10.21261846
4. Department of Health and Social Care. *Consultation Document: Changes to Human Medicine Regulations to Support the Rollout of COVID-19 Vaccines*; 2021. Accessed September 17, 2021. https://www.gov.uk/government/consultations/distributing-vaccines-and-treatments-for-covid-19-and-flu/consultation-document-changes-to-human-medicine-regulations-to-support-the-rollout-of-covid-19-vaccines#civil-liability-and-immunity
5. Chumakov K, Avidan MS, Benn CS, et al. Old vaccines for new infections: Exploiting innate immunity to control COVID-19 and prevent future pandemics. *Proceedings of the National Academy ofSciences of the United States of America*2021;118(21). doi:10.1073/pnas.2101718118
6. Government of Israel. *חיסון קבלת לאחר מאומתים קב בקרב ' הראשונים המחוסנים*. Accessed September 5, 2021. https://www.gov.il/BlobFolder/reports/vaccine-efficacy-safety-follow-up-committee/he/files_publications_corona_two-dose-vaccination-data.pdf
7. CDC COVID-19 Vaccine Breakthrough Case Investigations Team. COVID-19 Vaccine Breakthrough Infections Reported to CDC — United States, January 1–April 30, 2021. *Morbidity and Mortality Weekly Report*. 2021;70(21):792-793. Accessed September 15, 2021. https://www.cdc.gov/mmwr/volumes/70/wr/pdfs/mm7021e3-H.pdf
8. van Vinh Chau N, My Ngoc N, Anh Nguyet L, et al. *Transmission of SARS-CoV-2 Delta Variant among Vaccinated2 Healthcare Workers, Vietnam*. https://ssrn.com/abstract=3897733
9. Riemersma KK, Grogan BE, Kita-Yarbro A, et al. Vaccinated and unvaccinated individuals have similar viral loads in communities with a high prevalence of the SARS-CoV-2 delta variant. doi:10.1101/2021.07.31.21261387
10. Pouwels KB, Pritchard E, Matthews PC, et al. Impact of Delta on viral burden and vaccine effectiveness against new SARS-CoV-2 infections in the UK. *medRxiv: the preprint server for health sciences*. Published online August 24, 2021. doi:10.1101/2021.08.18.21262237
11. Badgujar KC, Badgujar VC, Badgujar SB. Vaccine development against coronavirus (2003 to present): An overview, recent advances, current scenario, opportunities and challenges. *Diabetes and Metabolic Syndrome: Clinical Research and Reviews*. 2020;14(5):1361-1376. doi:10.1016/j.dsx.2020.07.022
12. Cross R. Will the coronavirus help mRNA and DNA vaccines prove their worth? Chemical & Engineering News. Published April 3, 2020. Accessed September 16, 2021. https://cen.acs.org/pharmaceuticals/vaccines/coronavirus-help-mRNA-DNA-vaccines/98/i14
13. International Federation of Pharmaceutical Manufacturers & Associations (IFPMA). *The Complex Journey of aVaccine: The Steps Behind Developing a New Vaccine.*; 2019. Accessed September 16, 2021. https://www.ifpma.org/wp-content/uploads/2019/07/IFPMA-ComplexJourney-2019_FINAL.pdf
14. Mull NK, Mitchell MD, Brennan PJ. *Adverse Effects of Messenger RNA Vaccines: An Evidence Review from the Penn Medicine Center for Evidence-Based Practice December 2020.*; 2020. Accessed September 17, 2021. http://www.uphs.upenn.edu/cep/COVID/mRNA%20vaccine%20review%20final.pdf
15. Moderna Inc. Moderna Provides Clinical and Supply Updates on COVID-19 Vaccine Program Ahead of 2nd Annual Vaccines Day. Published April 2021. Accessed August 29, 2021. https://investors.modernatx.com/news-releases/news-release-details/moderna-provides-clinical-and-supply-updates-covid-19-vaccine
16. Baden LR, el Sahly HM, Essink B, et al. Efficacy and Safety of the mRNA-1273 SARS-CoV-2 Vaccine. *New England Journal of Medicine* 2021;384(5):403-416. doi:10.1056/nejmoa2035389
17. Thomas SJ, Moreira Jr ED, Kitchin N, et al. Six Month Safety and Efficacy of the BNT162b2 mRNA COVID-19 Vaccine. *medRxiv: the preprint server for health sciences*. Published online July 28, 2021. doi:10.1101/2021.07.28.21261159
18. Brown RB. Outcome Reporting Bias in COVID-19 mRNA Vaccine Clinical Trials. *Medicina (Lithuania)*. 2021;57(3):1-9. doi:10.3390/medicina57030199
19. Classen JB. US COVID-19 Vaccines Proven to Cause More Harm than Good Based on Pivotal Clinical Trial Data Analyzed Using the Proper Scientific Endpoint, "All Cause Severe Morbidity." *Trends Int Med*. 2021;1(1):1-6. Accessed September 17, 2021. https://www.scivisionpub.com/pdfs/us-covid19-vaccines-proven-to-cause-more-harm-than-good-based-on-pivotal-clinical-trial-data-analyzed-using-the-proper-scientific--1811.pdf
20. Grenfell BT, Pybus OG, Gog JR, et al. Unifying the Epidemiological and Evolutionary Dynamics of Pathogens. *Science*. 2004;303(5656):327-332. doi:10.1126/science.1090727
21. Sanjuán R, Domingo-Calap P. Mechanisms of viral mutation. *Cellular and Molecular Life Sciences*. 2016;73(23):4433-4448. doi:10.1007/s00018-016-2299-6
22. Moya AS, Elena SF, Bracho A, Miralles R, Barrio E. The evolution of RNA viruses: A population genetics view RNA Viruses: Biological and Population Properties. *PNAS*. 2000;97(13):6967-6973. doi:10.1073/pnas.97.13.6967
23. Elena SF, Sanjuán R. Adaptive Value of High Mutation Rates of RNA Viruses: Separating Causes from Consequences.*Journal of Virology* 2005;79(18):11555-11558. doi:10.1128/jvi.79.18.11555-11558.2005
24. Lauring AS, Andino R. Quasispecies Theory and the Behavior of RNA Viruses. *PLoS Pathogens*. 2010;6(7):1-8. doi:10.1371/journal.ppat.1001005
25. Schneider WL, Roossinck MJ. Genetic Diversity in RNA Virus Quasispecies Is Controlled by Host-Virus Interactions. *Journal of Virology* 2001;75(14):6566-6571. doi:10.1128/jvi.75.14.6566-6571.2001

26. Haddad D, John SE, Mohammad A, et al. SARS-CoV-2: Possible recombination and emergence of potentially more virulent strains. *PLoS ONE*. 2021;16(5). doi:10.1371/journal.pone.0251368
27. Carrasco-Hernandez R, Jácome R, Vidal YL, de León SP. Are RNA viruses candidate agents for the next global pandemic? A review. *ILAR Journal*. 2017;58(3):343-358. doi:10.1093/ilar/ilx026
28. Domingo E, Escarmís C, Sevilla N, et al. Basic concepts in RNA virus evolution. *The FASEB Journal*. 1996;10(8):859-864. doi:10.1096/fasebj.10.8.8666162
29. Jary A, Leducq V, Malet I, et al. Evolution of viral quasispecies during SARS-CoV-2 infection. *Clinical Microbiology and Infection*. 2020;26(11):1560.e1-1560.e4. doi:10.1016/j.cmi.2020.07.032
30. Armero A, Berthet N, Avarre JC. Intra-Host Diversity of SARS-Cov-2 Should Not Be Neglected: Case of the State of Victoria, Australia.*Viruses*. 2021;13(1). doi:10.3390/v13010133
31. Sousa A. mRNA, Nanolipid Particles and PEG: A Triad Never Used in Clinical Vaccines is Going to Be Tested on Hundreds of Millions of People. *Biomedical Journal of Scientific & TechnicalResearch*. 2021;34(1). doi:10.26717/bjstr.2021.34.005501
32. Lei Y, Zhang J, Schiavon CR, et al. SARS-CoV-2 Spike Protein Impairs Endothelial Function via Downregulation of ACE 2. *Circulation Research*. Published online 2021:1323-1326. doi:10.1161/CIRCRESAHA.121.318902
33. Blann AD. How a Damaged Blood Vessel Wall Contributes to Thrombosis and Hypertension. *Pathophysiology of Haemostasis and Thrombosis*. 2003;33(5-6):445-448. doi:10.1159/000083843
34. Rhea EM, Logsdon AF, Hansen KM, et al. The S1 protein of SARS-CoV-2 crosses the blood–brain barrier in mice. *Nature Neuroscience*. 2021;24(3):368-378. doi:10.1038/s41593-020-00771-8
35. Zhang L, Zhou L, Bao L, et al. SARS-CoV-2 crosses the blood-brain barrier accompanied with basement membrane disruption without tight junctions alteration. *Signal transduction and targeted therapy*. 2021;6(1):337. doi:10.1038/s41392-021-00719-9
36. Buzhdygan TP, Deore BJ, Baldwin-Leclair A, et al. The SARS-CoV-2 spike protein alters barrier function in 2D static and 3D microfluidic in-vitro models of the human blood-brain barrier. *bioRxiv: the preprint server for biology*. Published online June 16, 2021. doi:10.1101/2020.06.15.150912
37. Abbott NJ. Astrocyte-endothelial interactions and blood-brain barrier permeability. *J Anat*. 2002;200:629-638. doi:10.1046/j.1469-7580.2002.00064.x
38. Vargas G, Medeiros Geraldo LH, Gedeão Salomão N, Viana Paes M, Regina Souza Lima F, Carvalho Alcantara Gomes F. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and glial cells: Insights and perspectives. *Brain, Behavior, & Immunity - Health*. 2020;7:100127. doi:10.1016/j.bbih.2020.100127
39. Chen C-Y, Chou Y-C, Hsueh Y-P. SARS-CoV-2 D614 and G614 spike variants impair neuronal synapses and exhibit differential fusion ability. *bioRxiv: the preprint server for biology*. Published online December 3, 2020. doi:10.1101/2020.12.03.409763
40. Kase Y, Okano H. Neurological pathogenesis of SARS-CoV-2 (COVID-19): from virological features to clinical symptoms. *Inflammation and Regeneration*. 2021;41(1). doi:10.1186/s41232-021-00165-8
41. Puelles V, Lütgehetmann M, Lindenmeyer M, Sperhake J, Wong M. Multiorgan and Renal Tropism of SARS-CoV-2. *New England Journal of Medicine*. 2020;383(6):590-592. doi:10.1056/NEJMc2011400
42. Li MY, Li L, Zhang Y, Wang XS. Expression of the SARS-CoV-2 cell receptor gene ACE2 in a wide variety of human tissues.*Infectious Diseases of Poverty*. 2020;9(1). doi:10.1186/s40249-020-00662-x
43. Raghavan S, Kenchappa DB, Leo MD. SARS-CoV-2 Spike Protein Induces Degradation of Junctional Proteins That Maintain Endothelial Barrier Integrity. *Frontiers in Cardiovascular Medicine*. 2021;8. doi:10.3389/fcvm.2021.687783
44. Pallesen J, Wang N, Corbett KS, et al. Immunogenicity and structures of a rationally designed prefusion MERS-CoV spike antigen. *Proceedings of the National Academy of Sciences of the United States of America*2017;114(35):E7348-E7357. doi:10.1073/pnas.1707304114
45. Martínez-Flores D, Zepeda-Cervantes J, Cruz-Reséndiz A, Aguirre-Sampieri S, Sampieri A, Vaca L. SARS-CoV-2 Vaccines Based on the Spike Glycoprotein and Implications of New Viral Variants. *Frontiers in Immunology*. 2021;12. doi:10.3389/fimmu.2021.701501
46. Koenig P-A, Schmidt FI. Spike D614G — A Candidate Vaccine Antigen Against Covid-19. *New England Journal of Medicine* 2021;384(24):2349-2351. doi:10.1056/nejmcibr2106054
47. Corbett KS, Edwards DK, Leist SR, et al. SARS-CoV-2 mRNA vaccine design enabled by prototype pathogen preparedness. *Nature*. 2020;586:567-571. doi:10.1038/s41586-020-2622-0
48. Watanabe Y, Mendonça L, Allen ER, et al. Native-like SARS-CoV-2 Spike Glycoprotein Expressed by ChAdOx1 nCoV-19/AZD1222 Vaccine. *ACS Central Science*. 2021;7(4):594-602. doi:10.1021/acscentsci.1c00080
49. Wrapp D, Wang N, Corbett KS, et al. Cryo-EM structure of the 2019-nCoV spike in the prefusion conformation. *Science*. 2020;367(6483):1260-1263. doi:10.1126/science.abb2507
50. Ogata AF, Cheng C-A, Desjardins M, et al. Circulating Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Vaccine Antigen Detected in the Plasma of mRNA-1273 Vaccine Recipients. *Clinical Infectious Diseases*. Published online May 20, 2021. doi:10.1093/cid/ciab465
51. Wisnewski A v., Luna JC, Redlich CA. Human IgG and IgA responses to COVID-19 mRNA vaccines. *PLoS ONE*. 2021;16(6 June). doi:10.1371/journal.pone.0249499
52. Letarov A v., Babenko V v., Kulikov EE. Free SARS-CoV-2 Spike Protein S1 Particles May Play a Role in the Pathogenesis of COVID-19 Infection. *Biochemistry (Moscow)*. 2021;86(3):257-261. doi:10.1134/S0006297921030032
53. Yu J, Yuan X, Chen H, Chaturvedi S, Braunstein EM, Brodsky RA. Direct activation of the alternative complement pathway by SARS-CoV-2 spike proteins is blocked by factor D inhibition. *Blood*. 2020;136(18):2080-2089. http://ashpublications.org/blood/article-pdf/136/18/2080/1779273/bloodbld2020008248.pdf
54. Alto NM, Orth K. Subversion of Cell Signaling by Pathogens. *Cold Spring Harbor Perspectives in Biology*. 2012;4(9). doi:10.1101/cshperspect.a006114
55. Crowley SD, Gurley SB, Herrera MJ, et al. Angiotensin II causes hypertension and cardiac hypertrophy through its receptors in the kidney. *PNAS*. 2006;103(47):17985-17990. doi:10.1073/pnas.0605545103
56. Young CN, Davisson RL. Angiotensin-II, the brain, and hypertension: An update. *Hypertension*. 2015;66(5):920-926. doi:10.1161/HYPERTENSIONAHA.115.03624
57. Suzuki YJ, Gychka SG. SARS-CoV-2 Spike Protein Elicits Cell Signaling in Human Host Cells: Implications for Possible Consequences of COVID-19 Vaccines. *Vaccines*. 2021;9(1):1-8. doi:10.3390/vaccines9010036

58. Silvagno F, Vernone A, Pescarmona GP. The role of glutathione in protecting against the severe inflammatory response triggered by covid-19. *Antioxidants*. 2020;9(7):1-16. doi:10.3390/antiox9070624
59. Pfizer Inc. *SARS-CoV-2 MRNA Vaccine (BNT162, PF-07302048) 2.6.4* 薬物動態試験の概要文. Accessed August 29, 2021. https://www.pmda.go.jp/drugs/2021/P20210212001/672212000_30300AMX00231_I100_1.pdf
60. Pardi N, Tuyishime S, Muramatsu H, et al. Expression kinetics of nucleoside-modified mRNA delivered in lipid nanoparticles to mice by various routes. *Journal of Controlled Release* 2015;217:345-351. doi:10.1016/j.jconrel.2015.08.007
61. Angeli F, Spanevello A, Reboldi G, Visca D, Verdecchia P. SARS-CoV-2 vaccines: Lights and shadows. *European Journal of Internal Medicine* 2021;88:1-8. doi:10.1016/j.ejim.2021.04.019
62. Su JR. *Myopericarditis Following COVID-19 Vaccination: Updates from the Vaccine Adverse Event Reporting System (VAERS)*.; 2021. Accessed September 4, 2021. https://www.cdc.gov/vaccines/acip/meetings/downloads/slides-2021-08-30/03-COVID-Su-508.pdf
63. Beth T, Krug A, Stevenson J, Mandrola J. SARS-CoV-2 mRNA Vaccination-Associated Myocarditis in Children Ages 12-17: A Stratified National Database Analysis. Published online 2021. doi:10.1101/2021.08.30.21262866
64. Alban S. The "precautionary principle" as a guide for future drug development. *Eur J Clin Invest*. 2005;35(I):33-44. doi:10.1111/j.0960-135X.2005.01455.x
65. Hassett KJ, Higgins J, Woods A, et al. Impact of lipid nanoparticle size on mRNA vaccine immunogenicity. *Journal of Controlled Release* 2021;335:237-246. doi:10.1016/j.jconrel.2021.05.021
66. MHRA. *SpikeVax: Summary of Product Characteristics* Accessed September 5, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1012618/mhra-smpc-plgb-53720-amended_18-08_clean.pdf
67. Kumari S, Mg S, Mayor S. Endocytosis unplugged: Multiple ways to enter the cell. *Cell Research*. 2010;20(3):256-275. doi:10.1038/cr.2010.19
68. Hamming I, Timens W, Bulthuis MLC, Lely AT, Navis GJ, van Goor H. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis. *Journal of Pathology* 2004;203(2):631-637. doi:10.1002/path.1570
69. Ricard N, Bailly S, Guignabert C, Simons M. The quiescent endothelium: signalling pathways regulating organ-specific endothelial normalcy. *Nature Reviews Cardiology*. 2021;18(8):565-580. doi:10.1038/s41569-021-00517-4
70. Maugeri M, Nawaz M, Papadimitriou A, et al. Linkage between endosomal escape of LNP-mRNA and loading into EVs for transport to other cells. *Nature Communications*. 2019;10(1). doi:10.1038/s41467-019-12275-6
71. Zhang S, Liu Y, Wang X, et al. SARS-CoV-2 binds platelet ACE2 to enhance thrombosis in COVID-19. *Journal of Hematology and Oncology* 2020;13(1). doi:10.1186/s13045-020-00954-7
72. MHRA. *Summary of the Public Assessment Reportfor COVID-19 Vaccine Pfizer/BioNTech*.; 2021. Accessed September 12, 2021. https://www.gov.uk/government/publications/regulatory-approval-of-pfizer-biontech-vaccine-for-covid-19/summary-public-assessment-report-for-pfizerbiontech-covid-19-vaccine
73. Committee for Medicinal Products for Human Use (CHMP). *Committee for Medicinal Products for Human Use (CHMP) Assessment Report Assessment Report: Comirnaty Common Name: COVID-19 MRNA Vaccine (Nucleoside-Modified)*.; 2021. Accessed September 13, 2021. https://www.ema.europa.eu/en/documents/assessment-report/comirnaty-epar-public-assessment-report_en.pdf
74. Guevara ML, Persano F, Persano S. Advances in Lipid Nanoparticles for mRNA-Based Cancer Immunotherapy. *Frontiers in Chemistry*. 2020;8. doi:10.3389/fchem.2020.589959
75. World Health Organization. *Evaluation of theQuality, Safety and Efficacy of RNA-Based Prophylactic Vaccines for Infectious Diseases: Regulatory Considerations*.; 2020. Accessed September 17, 2021. https://www.who.int/docs/default-source/biologicals/ecbs/reg-considerations-on-rna-vaccines_1st-draft_pc_tz_22122020.pdf?sfvrsn=c13e1e20_3
76. Cui S, Wang Y, Gong Y, et al. Correlation of the cytotoxic effects of cationic lipids with their headgroups. *Toxicology Research*. 2018;7(3):473-479. doi:10.1039/c8tx00005k
77. Ura T, Okuda K, Shimada M. Developments in viral vector-based vaccines. *Vaccines*. 2014;2(3):624-641. doi:10.3390/vaccines2030624
78. *Reg 174 Information for UK Healthcare Professionals: COVID-19 Vaccine AstraZeneca, Solution for Injection, COVID-19 Vaccine (ChAdOx1-S [Recombinant])*.; 2021. Accessed September 6, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1012661/uk-spc-covid-19-vaccine-astrazeneca-reg174-gbs-clean.pdf
79. MHRA. *Public Assessment Report National Procedure: Vaxzevria (Previously COVID-19 Vaccine AstraZeneca, Suspension for Injection) COVID-19 Vaccine (ChAdOx1-S [Recombinant]) PLGB 17901/0355 AstraZeneca UK Limited*.; 2021. Accessed September 13, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1003840/CMA_UKPAR_COVID_19_Vaccine_AstraZeneca_PAR_16.07.2021.pdf
80. Rzymski P, Perek B, Flisiak R. Thrombotic Thrombocytopenia after COVID-19 Vaccination: In Search of the Underlying Mechanism. *Vaccines*. 2021;9(6). doi:10.3390/vaccines9060559
81. Diamond MS, Pierson TC. The Challenges of Vaccine Development against a New Virus during a Pandemic. *Cell Host and Microbe*. 2020;27(5):699-703. doi:10.1016/j.chom.2020.04.021
82. McFadyen JD, Stevens H, Peter K. The Emerging Threat of (Micro)Thrombosis in COVID-19 and Its Therapeutic Implications. *Circulation Research*. 2020;127(4):571-587. doi:10.1161/CIRCRESAHA.120.317447
83. *Public Assessment Report Authorisation for Temporary Supply COVID-19 MRNA Vaccine BNT162b2 (BNT162b2 RNA) Concentrate for Solution for Injection Annex 1 - Extension of Indication* Accessed September 5, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/997584/COVID-19_mRNA_Vaccine_BNT162b2__UKPAR___PFIZER_BIONTECH_ext_of_indication_11.6.2021.pdf
84. *A PHASE 1/2/3, PLACEBO-CONTROLLED Title: A Phase 1/2/3 Study to Evaluate the Safety, Tolerability, Immunogenicity, and Efficacy of RNA Vaccine Candidates Against COVID-19 in Healthy Individuals*.; 2020. Accessed September 6, 2021. https://cdn.pfizer.com/pfizercom/2020-11/C4591001_Clinical_Protocol_Nov2020.pdf

85. Food and Drug Administration (FDA). *Emergency Use Authorization (EUA) for an Unapproved Product Review Memorandum Identifying Information Application Type EUA (Event-Driven EUA Request) Application Number 27034 Sponsor Pfizer, Inc., on Behalf of Pfizer and BioNTech Submission Date*; 2020. Accessed September 17, 2021. https://www.fda.gov/media/144416/download
86. WhatDoTheyKnow. Full nucleotide base sequences for all of the COVID vaccines approved by the MRHA. Accessed September 12, 2021. https://www.whatdotheyknow.com/request/full_nucleotide_base_sequences_f_2?fbclid=IwAR3oxXzmU2JNWUtQnVCj-sN77mUgB8myz0G2eztDZs9JHrClv_-aw88ry9A
87. Vojdani A, Kharrazian D. Potential antigenic cross-reactivity between SARS-CoV-2 and human tissue with a possible link to an increase in autoimmune diseases. *Clinical Immunology*. 2020;217. doi:10.1016/j.clim.2020.108480
88. Doulberis M, Papaefthymiou A, Kotronis G, et al. Does COVID-19 Vaccination Warrant the Classical Principle "ofelein i mi vlaptin"? *medicina*. 2021;57(253). doi:10.3390/medicina57030253
89. Dotan A, Kanduc D, Muller S, Makatsariya A, Shoenfeld Y. Molecular mimicry between SARS-CoV-2 and the female reproductive system. *Am J Reprod Immunol*. Published online August 18, 2021:1-8. doi:10.1111/aji.13494
90. Seneff S, Nigh G. Worse Than the Disease? Reviewing Some Possible Unintended Consequences of the mRNA Vaccines Against COVID-19. *International Journal of Vaccine Theory, Practice, and Research* 2021;2(1):38-79. Accessed September 5, 2021. https://dpbh.nv.gov/uploadedFiles/dpbhnvgov/content/Boards/BOH/Meetings/2021/SENEFF~1.PDF
91. Ehlinger C, Spear N, Doddareddy R, Shankar G, Schantz A. A generic method for the detection of polyethylene glycol specific IgG and IgM antibodies in human serum. *Journal of ImmunologicalMethods*. 2019;474. doi:10.1016/j.jim.2019.112669
92. Shiraishi K, Yokoyama M. Toxicity and immunogenicity concerns related to PEGylated-micelle carrier systems: a review. *Science and Technology of Advanced Materials*. 2019;20(1):324-336. doi:10.1080/14686996.2019.1590126
93. Moghimi SM. Allergic Reactions and Anaphylaxis to LNP-Based COVID-19 Vaccines. *Molecular Therapy*. 2021;29(3):898-900. doi:10.1016/j.ymthe.2021.01.030
94. Psichogiou M, Samarkos M, Mikos N, Hatzakis A. Reactivation of Varicella Zoster virus after Vaccination for SARS-CoV-2. *Vaccines*. 2021;9(6). doi:10.3390/vaccines9060572
95. Kanata E, Llorens F, Dafou D, et al. RNA editing alterations define manifestation of prion diseases. *Proceedings of the National Academy of Sciences of the United States of America* 2019;116(39):19727-19735. doi:10.1073/pnas.1803521116
96. Hou X, Zaks T, Langer R, Dong Y. Lipid nanoparticles for mRNA delivery. *Nature Reviews Materials*. Published online 2021. doi:10.1038/s41578-021-00358-0
97. Yetisgin AA, Cetinel S, Zuvin M, Kosar A, Kutlu O. Therapeutic nanoparticles and their targeted delivery applications. *Molecules*. 2020;25(9). doi:10.3390/molecules25092193
98. Zhang Y. Cell toxicity mechanism and biomarker. *Clinical and Translational Medicine*. 2018;7(1). doi:10.1186/s40169-018-0212-7
99. *Design and Analysis of Shedding Studies for Virusor Bacteria-Based Gene Therapy and Oncolytic Products - Guidance for Industry*.; 2015. Accessed September 6, 2021. https://www.fda.gov/files/vaccines%2C%20blood%20%26%20biologics/published/Design-and-Analysis-of-Shedding-Studies-for-Virus-or-Bacteria-Based-Gene-Therapy-and-Oncolytic-Products--Guidance-for-Industry.pdf
100. Walach H, Klement RJ, Aukema W. The Safety of COVID-19 Vaccinations—We Should Rethink the Policy. *Vaccines*. 2021;9(7). doi:10.3390/vaccines9070693
101. Vaccines Editorial Office. Retraction Walach et al. The Safety of COVID-19 Vaccinations We Should Rethink the Policy. Vaccines 2021, 9, 693. *Vaccines*. 2021;9(7):729. doi:10.3390/vaccines9070729
102. gov.uk. *Coronavirus (COVID-19) in the UK*. Accessed September 5, 2021. https://coronavirus.data.gov.uk/details/deaths
103. Bentley PM. Error rates in SARS-CoV-2 testing examined with Bayes' theorem. *Heliyon*. 2021;7(4). doi:10.1016/j.heliyon.2021.e06905
104. Rose J. A Report on the U.S. Vaccine Adverse Events Reporting System (VAERS) of the COVID-1 9 Messenger Ribonucleic Acid (mRNA) Biologicals. *Science, Public Health Policy, and The Law*. 2021;3:59-80. https://www.nia.nih.gov/sites/default/files/2018-09/nia-ae-and-sae-guidelines-2018.pdf
105. Sencer DJ, Millar JD. Reflections on the 1976 swine flu vaccination program. *Emerg Infect Dis*. 2006;12(1):29-33. doi:10.3201/eid1201.051007
106. Swine Flu: 60 Minutes with Mike Wallace. CBS. Accessed September 16, 2021. https://www.youtube.com/watch?v=lBV8VlGBkMQ
107. Neustadt RE, Fineberg H v. *Swine Flu Affair: Decision-Making on a Slippery Disease*. National Academy of Sciences; 1978. Accessed September 16, 2021. https://www.ncbi.nlm.nih.gov/books/NBK219606/pdf/Bookshelf_NBK219606.pdf
108. Weber B. David J. Sencer Dies at 86; Led Disease-Control Agency. *New York Times*. https://www.nytimes.com/2011/05/04/us/04sencer.html. Published May 3, 2011. Accessed September 16, 2021.
109. Patel SU, Khurram R, Lakhani A, Quirk B. Guillain-Barre syndrome following the first dose of the chimpanzee adenovirus-vectored COVID-19 vaccine, ChAdOx1. *BMJ Case Reports*. 2021;14(4). doi:10.1136/bcr-2021-242956
110. Medicines & Healthcare products Regulatory Agency (MHRA). Coronavirus vaccine - weekly summary of Yellow Card reporting. Published September 16, 2021. https://www.gov.uk/government/publications/coronavirus-covid-19-vaccine-adverse-reactions/coronavirus-vaccine-summary-of-yellow-card-reporting
111. European Medicines Agency. Meeting highlights from the Pharmacovigilance Risk Assessment Committee (PRAC) 5-8 July 2021. Published July 9, 2021. Accessed September 14, 2021. https://www.ema.europa.eu/en/news/meeting-highlights-pharmacovigilance-risk-assessment-committee-prac-5-8-july-2021
112. VAERS. VAERS Covid-19 Data. Accessed September 17, 2021. https://wonder.cdc.gov/vaers.html
113. Ciccone A. SARS-CoV-2 vaccine-induced cerebral venous thrombosis. *European Journal of Internal Medicine* 2021;89:19-21. doi:10.1016/j.ejim.2021.05.026
114. Chen W, Pan JY. Anatomical and Pathological Observation and Analysis of SARS and COVID-19: Microthrombosis Is the Main Cause of Death. *Biological Procedures Online*. 2021;23(1). doi:10.1186/s12575-021-00142-y
115. Wu F, Yan R, Liu M, et al. Antibody-dependent enhancement (ADE) of SARS-CoV-2 infection in recovered COVID-19 patients: studies based on cellular and structural biology analysis. *medRxiv: the preprint server for health sciences*. Published online October 13, 2020. doi:10.1101/2020.10.08.20209114

116. Ricke DO. Two Different Antibody-Dependent Enhancement (ADE) Risks for SARS-CoV-2 Antibodies. *Frontiers in Immunology*. 2021;12. doi:10.3389/fimmu.2021.640093
117. Pitts PJ. Implications of antibody-dependent enhancement of infection for SARS-CoV-2 countermeasures.*Nature Biotechnology*. 2020;38(7):788-789. doi:10.1038/s41587-020-0577-1
118. Amanat F, Thapa M, Lei T, et al. SARS-CoV-2 mRNA vaccination induces functionally diverse antibodies to NTD, RBD, and S2. *Cell*. 2021;184(15):3936-3948.e10. doi:10.1016/j.cell.2021.06.005
119. Yahi N, Chahinian H, Fantini J. Infection-enhancing anti-SARS-CoV-2 antibodies recognize both the original Wuhan/D614G strain and Delta variants. A potential risk for mass vaccination? *Journal of Infection* Published online August 2021. doi:10.1016/j.jinf.2021.08.010
120. Karthik K, Senthilkumar TMA, Udhayavel S, Raj GD. Role of antibody-dependent enhancement (ADE) in the virulence of SARS-CoV-2 and its mitigation strategies for the development of vaccines and immunotherapies to counter COVID-19. *Human Vaccines and Immunotherapeutics*. 2020;16(12):3055-3060. doi:10.1080/21645515.2020.1796425
121. Lee WS, Wheatley AK, Kent SJ, DeKosky BJ. Antibody-dependent enhancement and SARS-CoV-2 vaccines and therapies. *Nature Microbiology*. 2020;5(10):1185-1191. doi:10.1038/s41564-020-00789-5
122. Tseng C te, Sbrana E, Iwata-Yoshikawa N, et al. Immunization with SARS coronavirus vaccines leads to pulmonary immunopathology on challenge with the SARS virus. *PLoS ONE*. 2012;7(4). doi:10.1371/journal.pone.0035421
123. Liu L, Wei Q, Lin Q, et al. Anti-spike IgG causes severe acute lung injury by skewing macrophage responses during acute SARS-CoV infection. *JCI insight*. 2019;4(4). doi:10.1172/jci.insight.123158
124. Su S, Du L, Jiang S. Learning from the past: development of safe and effective COVID-19 vaccines. *Nature Reviews Microbiology*. 2021;19(3):211-219. doi:10.1038/s41579-020-00462-y
125. Bolles ME. Evaluations Of Severe Acute Respiratory Syndrome Coronavirus Therapeutics And A Viral Capacity For Plasticity And Escape. [PhD Thesis]. *University of North Carolina at Chapel Hill* Published online 2013. doi:10.17615/yqyj-ht59
126. Liu Y, Arase N, Kishikawa J-I, et al. The SARS-CoV-2 Delta variant is poised to acquire complete resistance to wild-type spike vaccines. *bioRxiv : the preprint server for biology*. Published online August 23, 2021. doi:10.1101/2021.08.22.457114
127. van Egeren D, Novokhodko A, Stoddard M, et al. Risk of rapid evolutionary escape from biomedical interventions targeting SARS-CoV-2 spike protein. *PloS one*. 2021;16(4):e0250780. doi:10.1371/journal.pone.0250780
128. Martin DP, Weaver S, Tegally H, et al. The emergence and ongoing convergent evolution of the N501Y lineages coincides with a major global shift in the SARS-CoV-2 selective landscape. *medRxiv: the preprint server for health sciences*. Published online March 5, 2021. doi:10.1101/2021.02.23.21252268
129. Park MS, Kim J il, Park S, Lee I, Park MS. Original Antigenic Sin Response to RNA Viruses and Antiviral Immunity. *Immune Network*. 2016;16(5):261-270. doi:10.4110/in.2016.16.5.261
130. Petráš M, Králová Lesná I. SARS-CoV-2 vaccination in the context of original antigenic sin. *Human Vaccines and Immunotherapeutics*. Published online 2021. doi:10.1080/21645515.2021.1949953
131. Brown EL, Essigmann HT, Anderson M, et al. Original Antigenic Sin: the Downside of Immunological Memory and Implications for COVID-19 HCoV-NL63 and HCoV-HKU1) (E. *American Society for Microbiology*. 2021;6(2):1-6. doi:10.1128/mSphere.00056-21
132. Keehner J, Horton LE, Binkin NJ, et al. Resurgence of SARS-CoV-2 Infection in a Highly Vaccinated Health System Workforce. *New England Journal of Medicine* Published online September 1, 2021. doi:10.1056/NEJMc2112981
133. Tada T, Zhou H, Samanovic MI, et al. Comparison of Neutralizing Antibody Titers Elicited by mRNA and Adenoviral Vector Vaccine against SARS-CoV-2 Variants. *bioRxiv: the preprint server for biology*. Published online July 21, 2021. doi:10.1101/2021.07.19.452771
134. Burugorri-Pierre C, Lafuente-Lafuente C, Oasi C, et al. Investigation of an Outbreak of COVID-19 in a French Nursing Home With Most Residents Vaccinated. *JAMA Network Open*. 2021;4(9):e2125294. doi:10.1001/jamanetworkopen.2021.25294
135. Public Health England. *COVID-19 Vaccine Surveillance Report: Week 36 (Published: 9 September 2021)*. Accessed September 17, 2021. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1016465/Vaccine_surveillance_report_-_week_36.pdf
136. Brown CM, Vostok J, Johnson H, et al. Outbreak of SARS-CoV-2 Infections, Including COVID-19 Vaccine Breakthrough Infections, Associated with Large Public Gatherings-Barnstable County, Massachusetts, July 2021. *MMWR Morb Mortal Wkly Rep*. 2021;70(31):1059-1062. https://www.cdc.gov/coronavirus/2019-ncov/community/large-events/
137. Suzuki YJ. The viral protein fragment theory of COVID-19 pathogenesis. *Medical Hypotheses*. 2020;144. doi:10.1016/j.mehy.2020.110267
138. Luo R, Delaunay-Moisan A, Timmis K, Danchin A. SARS-CoV-2 biology and variants: anticipation of viral evolution and what needs to be done. *Environmental Microbiology*. 2021;23(5):2339-2363. doi:10.1111/1462-2920.15487
139. Harvey WT, Carabelli AM, Jackson B, et al. SARS-CoV-2 variants, spike mutations and immune escape. *Nature Reviews Microbiology*. 2021;19(7):409-424. doi:10.1038/s41579-021-00573-0
140. Shrestha NK, Burke PC, Nowacki AS, Terpeluk P, Gordon SM. Necessity of COVID-19 vaccination in previously infected individuals. *medRxiv: the preprint server for health sciences*. Published online June 19, 2021. doi:10.1101/2021.06.01.21258176
141. Gazit S, Shlezinger R, Perez G, et al. Comparing SARS-CoV-2 natural immunity to vaccine-induced immunity: reinfections versus breakthrough infections. *medRxiv: the preprint server for health sciences*. Published online August 25, 2021. doi:10.1101/2021.08.24.21262415
142. Egbert ER, Xiao S, Colantuoni E, et al. Durability of Spike Immunoglobin G Antibodies to SARS-CoV-2 Among Health Care Workers With Prior Infection. *JAMA Network Open*. 2021;4(8):e2123256. doi:10.1001/jamanetworkopen.2021.23256
143. Rebholz H, Braun RJ, Saha T, Harzer O, Schneider M, Ladage D. Longitudinal monitoring of SARS-CoV-2-specific immune responses. *medRxiv: the preprint server for health sciences*. Published online August 20, 2021. doi:10.1101/2021.08.14.21262042
144. Assis R, Jain A, Nakajima R, et al. Distinct SARS-CoV-2 Antibody Responses Elicited by Natural Infection and mRNA Vaccination. *bioRxiv: the preprint server for biology*. Published online May 19, 2021. doi:10.1101/2021.04.15.440089
145. Ferretti AP, Kula T, Wang Y, et al. Unbiased Screens Show CD8+ T Cells of COVID-19 Patients Recognize Shared Epitopes in SARS-CoV-2 that Largely Reside outside the Spike Protein. *Immunity*. 2020;53(5):1095-1107.e3. doi:10.1016/j.immuni.2020.10.006
146. Cohen KW, Linderman SL, Moodie Z, et al. Longitudinal analysis shows durable and broad immune memory after SARS-CoV-2 infection with persisting antibody responses and memory B and T cells. *Cell Reports Medicine*. 2021;2(7). doi:10.1016/j.xcrm.2021.100354

147. Bertoletti A, le Bert N, Qui M, Tan AT. SARS-CoV-2-specific T cells in infection and vaccination. *Cellular & Molecular Immunology*. Published online September 1, 2021. doi:10.1038/s41423-021-00743-3
148. le Bert N, Tan AT, Kunasegaran K, et al. SARS-CoV-2-specific T cell immunity in cases of COVID-19 and SARS, and uninfected controls. *Nature*. 2020;584(7821):457-462. doi:10.1038/s41586-020-2550-z
149. Konstantin Föhse F, Geckin B, Overheul GJ, et al. The BNT162b2 mRNA vaccine against SARS-CoV-2 reprograms both adaptive and innate immune responses. *medRxiv: the preprint server for health sciences*. Published online May 6, 2021. doi:10.1101/2021.05.03.21256520
150. Zaru R. Pattern recognition receptors (PRRs): Toll-like receptors. British Society for Immunology. Accessed September 6, 2021. https://www.immunology.org/public-information/bitesized-immunology/receptors-and-molecules/pattern-recognition-receptors-prrs
151. Loske J, Röhmel J, Lukassen S, et al. Pre-activated antiviral innate immunity in the upper airways controls early SARS-CoV-2 infection in children. *Nature Biotechnology*. Published online 2021. doi:10.1038/s41587-021-01037-9
152. Weisblum Y, Schmidt F, Zhang F, et al. Escape from neutralizing antibodies by SARS-CoV-2 spike protein variants. *eLife*. 2020;9:1. doi:10.7554/eLife.61312
153. Rella SA, Kulikova YA, Dermitzakis ET, Kondrashov FA. Rates of SARS-CoV-2 transmission and vaccination impact the fate of vaccine-resistant strains. *Scientific Reports*. 2021;11(1):15729. doi:10.1038/s41598-021-95025-3
154. Area FB, Servellita CV, Morris M-K, et al. Predominance of antibody-resistant SARS-CoV-2 variants in vaccine breakthrough cases from the San Francisco Bay Area, California. *medRxiv: the preprint server for health sciences*. Published online August 25, 2021. doi:10.1101/2021.08.19.21262139
155. Kustin T, Harel N, Finkel U, et al. Evidence for increased breakthrough rates of SARS-CoV-2 variants of concern in BNT162b2-mRNA-vaccinated individuals. *Nature Medicine*. 2021;27(8):1379-1384. doi:10.1038/s41591-021-01413-7
156. Mass vaccination during pandemic a historic blunder: Nobel laureate Luc Montagnier. Zee News. Published May 25, 2021. Accessed September 5, 2021. https://www.msn.com/en-in/news/world/mass-vaccination-during-pandemic-a-historic-blunder-nobel-laureate-luc-montagnier/ar-AAKmnJr
157. Oreshkova N, Molenaar RJ, Vreman S, et al. SARS-CoV-2 infection in farmed minks, the Netherlands, April and May 2020. *Eurosurveillance*. 2020;25(23). doi:10.2807/1560-7917.ES.2020.25.23.2001005
158. Mykytyn AZ, Lamers MM, Okba NMA, et al. Susceptibility of rabbits to SARS-CoV-2. *Emerging Microbes and Infections*. 2021;10(1):1-7. doi:10.1080/22221751.2020.1868951
159. Riemersma KK, Grogan BE, Kita-Yarbro A, et al. Shedding of Infectious SARS-CoV-2 Despite Vaccination. *medRxiv: the preprint server for health sciences*. Published online August 24, 2021. doi:10.1101/2021.07.31.21261387
160. Miller IF, Jessica Metcalf C. Vaccine-driven virulence evolution: Consequences of unbalanced reductions in mortality and transmission and implications for pertussis vaccines. *Journal of theRoyal Society Interface*. 2019;16(161). doi:10.1098/rsif.2019.0642
161. Read AF, Baigent SJ, Powers C, et al. Imperfect Vaccination Can Enhance the Transmission of Highly Virulent Pathogens. *PLoS Biology*. 2015;13(7). doi:10.1371/journal.pbio.1002198
162. Alenquer M, Ferreira F, Lousa D, et al. Signatures in SARS-CoV-2 spike protein conferring escape to neutralizing antibodies. Diamond MS, ed. *PLOS Pathogens*. 2021;17(8):e1009772. doi:10.1371/journal.ppat.1009772
163. Liu Y, Liu J, Plante KS, et al. The N501Y spike substitution enhances SARS-CoV-2 transmission. *bioRxiv: the preprint server for biology*. Published online March 9, 2021. doi:10.1101/2021.03.08.434499
164. Aleem A, Samad ABA, Slenker AK. *Emerging Variants of SARS-CoV-2 And Novel Therapeutics Against Coronavirus (COVID-19)* StatPearls Publishing; 2021. Accessed September 5, 2021. https://www.ncbi.nlm.nih.gov/books/NBK570580/
165. Kosugi Y, Wu J, Yamasoba D, et al. SARS-CoV-2 Lambda variant exhibits higher infectivity and immune resistance. *bioRxiv: the preprint server for biology*. doi:10.1101/2021.07.28.454085
166. Khateeb J, Li Y, Zhang H. Emerging SARS-CoV-2 variants of concern and potential intervention approaches. *Critical Care*. 2021;25(1). doi:10.1186/s13054-021-03662-x
167. Tay JH, Porter AF, Wirth W, Duchene S. The emergence of SARS-CoV-2 variants of concern is driven by acceleration of the evolutionary rate. *medRxiv: the preprint server for health sciences*. Published online August 31, 2021. doi:10.1101/2021.08.29.21262799
168. Sen SR, Sanders EC, Santos AM, et al. Evidence for Deleterious Antigenic Imprinting in SARS-CoV-2 Immune Response. *bioRxiv: the preprint server for biology*. doi:10.1101/2021.05.21.445201
169. Aydillo T, Rombauts A, Stadlbauer D, et al. Immunological imprinting of the antibody response in COVID-19 patients. *Nature Communications*. 2021;12(1). doi:10.1038/s41467-021-23977-1
170. Pickard A, Calverley BC, Chang J, et al. Discovery of re-purposed drugs that slow SARS-CoV-2 replication in human cells. Pekosz A, ed. *PLOS Pathogens*. 2021;17(9):e1009840. doi:10.1371/journal.ppat.1009840
171. Du Y, Shi R, Zhang Y, et al. A broadly neutralizing humanized ACE2-targeting antibody against SARS-CoV-2 variants. *Nature Communications*. 2021;12(1). doi:10.1038/s41467-021-25331-x
172. te Velthuis AJW, van den Worml SHE, Sims AC, Baric RS, Snijder EJ, van Hemert MJ. Zn^{2+} inhibits coronavirus and arterivirus RNA polymerase activity in vitro and zinc ionophores block the replication of these viruses in cell culture. *PLoS Pathogens*. 2010;6(11). doi:10.1371/journal.ppat.1001176
173. Derwand R, Scholz M. Does zinc supplementation enhance the clinical efficacy of chloroquine/hydroxychloroquine to win todays battle against COVID-19? *Medical Hypotheses*. 2020;142. doi:10.1016/j.mehy.2020.109815
174. Derwand R, Scholz M, Zelenko V. COVID-19 outpatients: early risk-stratified treatment with zinc plus low-dose hydroxychloroquine and azithromycin: a retrospective case series study. *International Journal of Antimicrobial Agents* 2020;56(6). doi:10.1016/j.ijantimicag.2020.106214
175. Wessels I, Rolles B, Rink L. The Potential Impact of Zinc Supplementation on COVID-19 Pathogenesis. *Frontiers in Immunology*. 2020;11. doi:10.3389/fimmu.2020.01712
176. McCullough PA, Alexander PE, Armstrong R, Arvinte C, Bain AF, Bartlett RP. Multifaceted highly targeted sequential multidrug treatment of early ambulatory high-risk SARS-CoV-2 infection (COVID-19). *Reviews in Cardiovascular Medicine*. 2020;21(4):517-530. doi:10.31083/J.RCM.2020.04.264
177. McCullough PA, Kelly RJ, Ruocco G, et al. Pathophysiological Basis and Rationale for Early Outpatient Treatment of SARS-CoV-2 (COVID-19) Infection. *American Journal of Medicine* 2021;134(1):16-22. doi:10.1016/j.amjmed.2020.07.003

178. Sanchez-Gonzalez MA, Moskowitz D, Issuree PD, Yatzkan G, Rizvi SAA, Day K. A Pathophysiological Perspective on COVID-19's Lethal Complication: From Viremia to Hypersensitivity Pneumonitis-like Immune Dysregulation. *Infection and Chemotherapy*. 2020;52(3):335-344. doi:10.3947/ic.2020.52.3.335
179. Yoshimura R, Katsuki A, Atake K, Hori H, Igata R, Konishi Y. Influence of fluvoxamine on plasma interleukin-6 or clinical improvement in patients with major depressive disorder. *Neuropsychiatric Disease and Treatment*. 2017;13:437-441. doi:10.2147/NDT.S123121
180. Rosen DA, Seki SM, Fernández-Castañeda A, et al. Modulation of the sigma-1 receptor-IRE1 pathway is beneficial in preclinical models of inflammation and sepsis. *Sci Transl Med*. 2019;11:5266. doi:10.1126/scitranslmed.aau5266
181. Hojyo S, Uchida M, Tanaka K, et al. How COVID-19 induces cytokine storm with high mortality. *Inflammation and Regeneration*. 2020;40(1). doi:10.1186/s41232-020-00146-3
182. Lenze EJ, Mattar C, Zorumski CF, et al. Fluvoxamine vs Placebo and Clinical Deterioration in Outpatients with Symptomatic COVID-19: A Randomized Clinical Trial. *JAMA - Journal of the AmericanMedical Association*. 2020;324(22):2292-2300. doi:10.1001/jama.2020.22760
183. Seftel D, Boulware DR. Prospective Cohort of Fluvoxamine for Early Treatment of Coronavirus Disease 19.*Open Forum Infectious Diseases*. 2021;8(2). doi:10.1093/ofid/ofab050
184. Crump A, Omura S. Ivermectin, "Wonder drug" from Japan: The human use perspective. *Proceedings of the Japan Academy Series B: Physical and Biological Sciences*. 2011;87(2):13-28. doi:10.2183/pjab.87.13
185. Rajter JC, Sherman MS, Fatteh N, Vogel F, Sacks J, Rajter JJ. Use of Ivermectin Is Associated With Lower Mortality in Hospitalized Patients With Coronavirus Disease 2019: The Ivermectin in COVID Nineteen Study. *Chest*. 2021;159(1):85-92. doi:10.1016/j.chest.2020.10.009
186. Zaidi AK, Dehgani-Mobaraki P. The mechanisms of action of Ivermectin against SARS-CoV-2: An evidence-based clinical review article.*Journal of Antibiotics* Published online 2021. doi:10.1038/s41429-021-00430-5
187. Lehrer S, Rheinstein PH. Ivermectin Docks to the SARS-CoV-2 Spike Receptor-binding Domain Attached to ACE2. *In Vivo*. 2020;34(5):3023-3026. doi:10.21873/invivo.12134
188. Santin AD, Scheim DE, McCullough PA, Yagisawa M, Borody TJ. Ivermectin: a multifaceted drug of Nobel prize-honoured distinction with indicated efficacy against a new global scourge, COVID-19. *New Microbes and New Infections*. 2021;43:100924. doi:10.1016/j.nmni.2021.100924
189. Morgenstern J, Redondo JN, Olavarria A, et al. Ivermectin as a SARS-CoV-2 Pre-Exposure Prophylaxis Method in Healthcare Workers: A Propensity Score-Matched Retrospective Cohort Study. *Cureus*. 2021;13(8). doi:10.7759/cureus.17455
190. Hossain KS, Hossain MG, Moni A, et al. Prospects of honey in fighting against COVID-19: pharmacological insights and therapeutic promises. *Heliyon*. 2020;6(12). doi:10.1016/j.heliyon.2020.e05798
191. Rahman MM, Mosaddik A, Alam AK. Traditional foods with their constituent's antiviral and immune system modulating properties. *Heliyon*. 2021;7(1). doi:10.1016/j.heliyon.2021.e05957
192. Joachimiak MP. Zinc against COVID-19? Symptom surveillance and deficiency risk groups. *PLoS Neglected Tropical Diseases*. 2021;15(1):1-17. doi:10.1371/journal.pntd.0008895
193. Szarpak L, Rafique Z, Gasecka A, et al. A systematic review and meta-analysis of effect of vitamin D levels on the incidence of COVID-19. *Cardiology Journal*. 2021;28(5):647-654. doi:10.5603/CJ.a2021.0072
194. Colunga Biancatelli RML, Berrill M, Catravas JD, Marik PE. Quercetin and Vitamin C: An Experimental, Synergistic Therapy for the Prevention and Treatment of SARS-CoV-2 Related Disease (COVID-19). *Frontiers in Immunology*. 2020;11. doi:10.3389/fimmu.2020.01451
195. Shi Z, Puyo CA. N-Acetylcysteine to Combat COVID-19: An Evidence Review. *Therapeutics and Clinical Risk Management*. 2020;16:1047-1055. doi:10.2147/TCRM.S273700
196. Mrityunjaya M, Pavithra V, Neelam R, Janhavi P, Halami PM, Ravindra P v. Immune-Boosting, Antioxidant and Anti-inflammatory Food Supplements Targeting Pathogenesis of COVID-19. *Frontiers in Immunology*. 2020;11. doi:10.3389/fimmu.2020.570122
197. Sallis R, Young DR, Tartof SY, et al. Physical inactivity is associated with a higher risk for severe COVID-19 outcomes: A study in 48 440 adult patients. *British Journal of Sports Medicine* 2021;55:1099-1105. doi:10.1136/bjsports-2021-104080
198. Skopec R. Coronavirus is a Biological Warfare Weapon. *Journal of Vaccines & Vaccination* 2021;12(2):1-6. doi:10.24966/CSMC-8801/100103
199. The White House Office of Science and Technology Policy and Department of Health and Human Services.*Doing Diligence to Assess the Risks and Benefits of Life Sciences Gain-of-Function Research*; 2014. Accessed September 15, 2021. https://obamawhitehouse.archives.gov/blog/2014/10/17/doing-diligence-assess-risks-and-benefits-life-sciences-gain-function-research
200. HHS. *U.S. Government Gain-of-Function Deliberative Process and Research Funding Pause on Selected Gain-of-Function Research Involving Influenza, MERS, and SARS Viruses*.; 2014. Accessed September 15, 2021. http://www.phe.gov/s3/dualuse/Documents/gain-of-function.pdf
201. National Institutes of Health (NIH). *Notice Announcing the Removal of the Funding Pause for Gain-of-Function Research Projects*; 2017. Accessed September 15, 2021. https://grants.nih.gov/grants/guide/notice-files/NOT-OD-17-071.html
202. U.S. Department of Health and Human Services. *Department of Health and Human Services Frameworkfor Guiding Funding Decisions about Proposed Research Involving Enhanced Potential Pandemic Pathogens*.; 2017. Accessed September 15, 2021. https://www.phe.gov/s3/dualuse/Documents/P3CO.pdf
203. McCaul MT. *The Origins of COVID-19: An Investigationof the Wuhan Institute of Virology*; 2021. Accessed September 16, 2021. https://gop-foreignaffairs.house.gov/wp-content/uploads/2021/08/ORIGINS-OF-COVID-19-REPORT.pdf
204. vanden Bossche G. PUBLIC HEALTH EMERGENCY OF INTERNATIONAL CONCERN: Why mass vaccination amidst a pandemic creates an irrepressible monster. Published March 6, 2021. Accessed September 6, 2021. https://37b32f5a-6ed9-4d6d-b3e1-5ec648ad9ed9.filesusr.com/ugd/28d8fe_266039aeb27a4465988c37adec9cd1dc.pdf
205. vanden Bossche G. Why should current Covid-19 vaccines not be used for mass vaccination during a pandemic? Published March 10, 2021. Accessed September 6, 2021. https://www.youtube.com/watch?v=shg0VWkz0VM
206. Pfizer Inc., BioNTech SE. Pfizer-BioNTech Announce Positive Topline Results of Pivotal COVID-19 Vaccine Study in Adolescents. Published March 31, 2021. Accessed September 6, 2021. https://www.pfizer.com/news/press-release/press-release-detail/pfizer-biontech-announce-positive-topline-results-pivotal
207. Pfizer Inc. Clinical Trials in Children. Accessed September 6, 2021. https://www.pfizer.com/science/clinical-trials/children
208. Centers for Disease Control and Prevention. Grading of Recommendations, Assessment, Development, and Evaluation (GRADE): Pfizer-BioNTech COVID-19 Vaccine for Persons Aged 12-15 Years. Accessed September 6, 2021. https://www.cdc.gov/vaccines/acip/recs/grade/covid-19-pfizer-biontech-vaccine-12-15-years.html#appendix02

209. Frenck RW, Klein NP, Kitchin N, et al. Safety, Immunogenicity, and Efficacy of the BNT162b2 Covid-19 Vaccine in Adolescents. *New England Journal of Medicine* 2021;385(3):239-250. doi:10.1056/nejmoa2107456

210. Sen. Ron Johnson with families on adverse reactions to COVID vaccine | FOX6 News Milwaukee [Video]. FOX6 News Milwaukee. Published June 28, 2021. Accessed September 6, 2021. https://www.youtube.com/watch?v=6mxqC9SiRh8

211. NewsRescue. #SilencedVoices: Vaccine-damaged Americans Tearfully Describe SEVERE Side Effects After Taking Covid Vaccine On Sen. Ron Johnson's 'Give an Ear' Panel. Published June 30, 2021. Accessed September 6, 2021. https://newsrescue.com/silencedvoices-vaccine-damaged-americans-tearfully-describe-severe-side-effects-after-taking-covid-vaccine-on-sen-ron-johnsons-give-an-ear-panel/