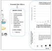
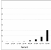
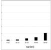
Outline

Highlights
Abstract
Graphical abstract
Keywords
1. Introduction
2. Background
3. Mass inoculation
4. Discussion
5. Overall conclusions
Author's contribution
Ethical approval
Declaration of Competing Interest
Acknowledgement
Appendix A
Appendix B
Appendix C
Appendix D
Appendix Da
References
Show full outline

Figures (4)

Tables (5)

⊞ Table 1
⊞ Table A1
⊞ Table
⊞ Table A2
⊞ Table A3

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Why are we vaccinating children against COVID-19?

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Erratum to "Why are we vaccinating children against COVID-19?" [Toxicol. Rep. 8C (202…
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Highlights

- Bulk of COVID-19 per capita deaths occur in elderly with high comorbidities.
- Per capita COVID-19 deaths are negligible in children.
- Clinical trials for these inoculations were very short-term.
- Clinical trials did not address long-term effects most relevant to children.
- High post-inoculation deaths reported in VAERS (very short-term).

Abstract

This article examines issues related to COVID-19 inoculations for children. The bulk of the official COVID-19-attributed deaths per capita occur in the elderly with high comorbidities, and the COVID-19 attributed deaths per capita are negligible in children. The bulk of the normalized post-inoculation deaths also occur in the elderly with high comorbidities, while the normalized post-inoculation deaths are small, but not negligible, in children. Clinical trials for these inoculations were very short-term (a few months), had samples not representative of the total population, and for adolescents/children, had poor predictive power because of their small size. Further, the clinical trials did not address changes in biomarkers that could serve as early warning indicators of elevated predisposition to serious diseases. Most importantly, the clinical trials did not address long-term effects that, if serious, would be borne by children/adolescents for potentially decades.

A novel *best-case scenario* cost-benefit analysis showed *very conservatively* that there are five times the number of deaths attributable to each inoculation vs those attributable to COVID-19 in the most vulnerable 65+ demographic. The risk of death from COVID-19 decreases drastically as age decreases, and the longer-term effects of the inoculations on lower age groups will increase their risk-benefit ratio, perhaps substantially.

Graphical abstract

Part of special issue:
SI: COVID-19 Pandemic: Health impact and Novel research
Edited by Konstantinos Poulas, Demetrios Kouretas, Aristides Tsatsakis, Konstantinos Farsalinos
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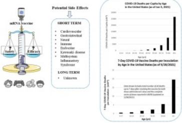
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Efficacy
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SHORT TERM
Cardiovascular
Gastrointestinal
Neural
Immune
Endocrine
Kawasaki disease
Multisystem Inflammatory Syndrome
LONG TERM
Unknown
COVID-19 Deaths per Capita by Age in the United States (as of Jun 5, 2021)
Age (yrs)
7-Day COVID-19 Vaccine Deaths per Inoculation by Age in the United States (as of 5/28/2021)
Age (yrs)

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Keywords

COVID-19; SARS-CoV-2; Inoculation; mRNA vaccines; Viral vector vaccines; Adverse events; Vaccine safety

1. Introduction

Currently, we are in the fifteenth month of the WHO-declared global COVID-19 pandemic. Restrictions of different severity are still in effect throughout the world [1]. The global COVID-19 mass inoculation is in its eighth month. As of this writing in mid-June 2021, over 800,000,000 people globally have received at least one dose of the inoculation and roughly half that number have been fully inoculated [2]. In the USA, about 170,000,000 people have received at least one dose and roughly 80 % of that number have been fully inoculated [2].

Also, in the USA, nearly 600,000 deaths have been officially attributed to COVID-19. Almost 5,000 deaths following inoculation have been reported to VAERS by late May 2021; specifically, “Over 285 million doses of COVID-19 vaccines were administered in the United States from December 14, 2020, through May 24, 2021. During this time, VAERS received 4,863 reports of death (0.0017 %) among people who received a COVID-19 vaccine.” [3] (the Vaccine Adverse Events Reporting System (VAERS) is a passive surveillance system managed jointly by the CDC and FDA [3]. Historically, VAERS has been shown to report about 1% of actual vaccine/inoculation adverse events [4]. See Appendix 1 for a first-principles confirmation of that result). By mid-June, deaths following COVID-19 inoculations had reached the ~6000 levels.

A vaccine is legally defined as any substance designed to be administered to a human being for the prevention of one or more diseases [5]. For example, a January 2000 patent application that defined vaccines as “compositions or mixtures that when introduced into the circulatory system of an animal will evoke a protective response to a pathogen.” was rejected by the U.S. Patent Office because “The immune response produced by a vaccine must be more than merely some immune response but must be protective. As noted in the previous Office Action, the art recognizes the term "vaccine" to be a compound which prevents infection” [6]. In the remainder of this article, we use the term ‘inoculated’ rather than vaccinated, because the injected material in the present COVID-19 inoculations prevents neither viral infection nor transmission. Since its main function in practice appears to be symptom suppression, it is operationally a “treatment”.

In the USA, inoculations were administered on a priority basis. Initially, first responders and frontline health workers, as well as the frailest elderly, had the highest priority. Then the campaign became more inclusive of lower age groups. Currently, approval has been granted for inoculation administration to the 12–17 years demographic, and the target for this demographic is to achieve the largest number of inoculations possible by the start of school in the Fall. The schedule for inoculation administration to the 5–11 years demographic has been accelerated to start somewhere in the second half of 2021, and there is the possibility that infants as young as six months may begin to get inoculated before the end of 2021 [7].

The remainder of this article will focus on the USA situation, and address mainly the pros and cons of inoculating children under eighteen. The article is structured as follows:

Section 1 (the present section) introduces the problem.

Section 2 (Background):
1) provides the background for the declared COVID-19 “pandemic” that led to

the present inoculations;

2) describes the clinical trials that provided the justification for obtaining Emergency Use Authorization (EUA) from the FDA to administer the inoculations to the larger population;
3) shows why the clinical trials did not predict either the seriousness of adverse events that have occurred so far (as reported in VAERS) or the potential extent of the underlying pre-symptomatic damage that has occurred as a result of the inoculations.

Section 3 (Mass Inoculation) summarizes the adverse events that have occurred already (through reporting in VAERS) from the mass inoculation and will present biological evidence to support the potential occurrence of many more adverse effects from these inoculations in the mid-and long-term.

Section 4 (Discussion) addresses these effects further

Section 5 (Summary and Conclusions) presents the conclusions of this study.

There are four appendices to this paper.

Appendix A provides some idea of the level of under-reporting of post-inoculation adverse events to VAERS and presents estimations of the actual number of post-inoculation deaths based on extrapolating the VAERS results to real-world experiences.

Appendix B provides a detailed analysis of the major clinical trials that were used to justify EUA for the inoculants presently being administered in the USA.

Appendix C summarizes potential adverse effects shown to have resulted from past vaccines, all of which could potentially occur as a result of the present inoculations.

Appendix D presents a novel *best-case scenario* cost-benefit analysis of the COVID-19 inoculations that have been administered in the USA.

2. Background

2.1. Pandemic history

In December 2019, a viral outbreak was reported in Wuhan, China, and the responsible coronavirus was termed Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [8,9]. The associated disease was called Coronavirus Disease 2019, or COVID-2019. The virus spread worldwide, and a global pandemic was declared by the WHO in March 2020 [10,11]. Restrictive measures of differing severity were implemented by countries globally, and included social distancing, quarantining, face masks, frequent hand sanitation, etc. [12,13]. In the USA, these measures were taken as well, differing from state-to-state [14]. At the same time, vaccine development was initiated to control COVID-19 [15]. In the USA, non-vaccine treatments were not encouraged at the Federal level, but different treatment regimens were pursued by some healthcare practitioners on an individual level [11,16,17].

By the end of May 2021, the official CDC death count attributed to COVID-19 was approaching 600,000, as stated previously. This number has been disputed for many reasons. First, before COVID-19 testing began, or in the absence of testing, after it was available, the diagnosis of COVID-19 (in the USA) could be made by the presumption of the healthcare practitioner that COVID-19 existed [4,18]. Second, after testing began, the main diagnostic used was the RT-PCR test. This test was done at very high amplification cycles, ranging up to 45 [[19], [20], [21]]. In this range, very high numbers of false positives are possible [22].

Third, most deaths attributed to COVID-19 were elderly with high comorbidities [1,22]. As we showed in a previous study [22], attribution of death to one of many possible comorbidities or especially toxic exposures in combinations [23] is highly arbitrary and can be viewed as a political decision more than a medical decision. For over 5 % of these deaths, COVID-19 was the only cause mentioned on the death certificate. For deaths with conditions or causes in addition to COVID-19, on average, there were 4.0 additional conditions or causes per death [24]. These deaths with comorbidities could equally have been ascribed to any of the comorbidities [22]. Thus, the actual number of COVID-19-based deaths in the USA may have been on the order of 35,000 or less, characteristic of a mild flu season.

Even the 35,000 deaths may be an overestimate. Comorbidities were based on the clinical definition of specific diseases, using threshold biomarker levels and relevant symptoms for the disease(s) of interest [25,26]. But many people have what are known as pre-clinical conditions. The biomarkers have not reached the threshold level for official disease diagnosis, but their abnormality reflects some degree of underlying dysfunction. The immune system response (including pre-clinical conditions) to the COVID-19 viral trigger should not be expected to be the same as the response of a healthy immune system [27]. If pre-clinical conditions had been taken into account and coupled with the false positives as well, the CDC estimate of 94 % misdiagnosis would be substantially higher.

2.2. Clinical trials

2.2.1. Clinical trials to gain FDA Emergency Use Authorization (EUA) approval

The unprecedented accelerated development of COVID-19 vaccines in the USA, dubbed Operation Warp Speed, resulted in a handful of substances available for clinical trials by mid-2020 [28]. These clinical trials were conducted to predict the safety and efficacy of the potential vaccines (which have turned out to be treatments/inoculations as stated previously), and thereby gain approval for inoculating the public at large [29]. An overview of the Pfizer clinical trials is presented in this section, and a more detailed description of the main clinical trials is shown in Appendix B.

Two types of inoculants have gained FDA EUA in the US: mRNA-based inoculants and viral vector-based inoculants, with the mRNA inoculants having the widest distribution so far. Comirnaty is the brand name of the mRNA-based inoculant developed by Pfizer/BioNTech, and Moderna COVID-19 Vaccine is the brand name of the mRNA-based inoculant developed by Moderna [30]. Both inoculants contain the genetic information needed for the production of the viral protein S (spike), which stimulates the development of a protective immune response against COVID-19 [31]. Janssen COVID-19 Vaccine is the brand name of the viral vector-based inoculant developed by Johnson and Johnson. Janssen COVID-19 vaccine uses an adenovirus to transport a gene from the coronavirus into human cells, which then produce the coronavirus spike protein. This spike protein primes the immune system to fight off potential coronavirus infection [32].

The results of these trials that allowed granting of EUA by the FDA can be found in the inserts to the inoculation materials. For example, the Pfizer inoculation trial results are contained in the fact sheet for healthcare providers administering vaccine (vaccination providers) [33].

There were two clinical trials conducted to gain FDA EUA for Pfizer: a smaller Phase 1/2 study, and a larger Phase 1/2/3 study. The age demographics for the larger clinical study are as follows (from the Pfizer insert): "Of the total number of Pfizer-BioNTech COVID-19 Vaccine recipients in Study 2 (N = 20,033), 21.4 % (n = 4,294) were 65 years of age and older and 4.3 % (n = 860) were 75 years of age and older." Additionally: "In an analysis of Study 2, based on data up to the cutoff date of March 13, 2021, 2,260 adolescents (1,131 Pfizer-BioNTech COVID-19 Vaccine; 1,129 placebo) were 12 through 15 years of age. Of these, 1,308 (660 Pfizer-BioNTech COVID-19 Vaccine and 648 placebo) adolescents have been followed for at least 2 months after the second dose of Pfizer-BioNTech COVID-19 Vaccine. The safety evaluation in Study 2 is ongoing."

The relevant demographics are presented in Table 7 on p.31 of the Pfizer insert. The age component of those demographics is shown below in Table 1.

Table 1. Demographics (population for the primary efficacy endpoint). The number of participants who received vaccine and placebo, stratified by age.

AGE GROUP	**Pfizer-BioNTech COVID-19 Vaccine (N = 18,242) n (%)**	**Placebo (N = 18,379) n (%)**
≥12 through 15 years[b]	46 (0.3 %)	42 (0.2 %)
≥16 through 17 years	66 (0.4 %)	68 (0.4 %)
≥16 through 64 years	14,216 (77.9 %)	14,299 (77.8 %)
≥65 through 74 years	3176 (17.4 %)	3226 (17.6 %)
≥75 years	804 (4.4 %)	812 (4.4 %)

Symbols: b: "100 participants 12 through 15 years of age with limited follow-up in the randomized population received at least one dose (49 in the vaccine group and 51 in the placebo group). Some of these participants were included in the efficacy evaluation depending on the population analyzed.

They contributed to exposure information but with no confirmed COVID-19 cases, and did not affect efficacy conclusions.", N: number of test subjects, n: number of controls.

There are very minor differences between most of the data in the above table and the preceding narrative shown, and they are probably due to different time horizons. The major difference is the number of adolescents used and appears to result from a much later reporting time.

Fig. 1 uses the official large CDC numbers (coupled with USA census data estimates from CDC Wonder) to show the COVID-19 deaths per capita as a function of age, circa early June 2021. Unfortunately, the most critical range, 85+, has the least resolution. It is obvious that most of the deaths occurred in the 55 to 100+ range, and the remaining individuals in the other ranges (especially under 35) have negligible risk of dying from the disease.

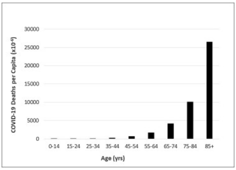

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Fig. 1. COVID-19 Deaths per capita by age in the United States (as of Jun 5, 2021). Population-based on U.S. CDC WONDER Bridge-Race Population Estimate 2019. Data obtained from https://wonder.cdc.gov/bridged-race-v2019.html on 6/15/2021. Provisional COVID-19 deaths based on CDC data provided by the National Center for Health Statistics for the period 1/1/2020 – 6/5/2021. Data obtained from https://data.cdc.gov/NCHS/Provisional-COVID-19-Deaths-by-Sex-and-Age/9bhg-hcku on 6/10/2021.

The age distribution in Fig. 1 differs substantially from the age distribution in Table 1. Why is this important? When designing a trial for the efficacy and safety of a potential treatment, the focus should be on the target population who could benefit from that treatment. There is little rationale for including participants in a trial for whom the treatment would not be relevant or warranted.

For the COVID-19 Pfizer trials, based on the data from Fig. 1, the trial population should have been limited at most to the 45–100+ age segment, appropriately weighted toward the higher end where the deaths per capita are most frequent. That was almost the exact opposite of what was done in the Pfizer clinical trials. In Fig. 1, approximately 58 % of the deaths occurred in the age range 75+, whereas 4.4 % of the participants in the Pfizer clinical trial were 75 + . Thus, the age range most impacted by COVID-19 deaths was minimally represented in the Pfizer clinical trials, and the age range least impacted by COVID-19 deaths was maximally represented in the Pfizer clinical trials. This skewed sampling has major implications for predicting the expected numbers of deaths for the target population from the clinical trials.

Besides age, the other metric of importance in determining COVID-19 deaths is the presence of comorbidities. The more comorbidities, and the more severe the comorbidities, the greater the chances of death or severe adverse outcomes from COVID-19. It is not clear how well the number and severity of comorbidities in the clinical trial sample matched those reflected in Fig. 1, but the insert does mention the large number of conditions that excluded participation in the trials. In sum, the results from the clinical trials could not be expected to reflect the results that could occur (and have occurred) from mass inoculation of the public, given the unaffected nature of the bulk of the trial population from SARS-CoV-2 exposure.

The prior discussion on the clinical trials has focused on the efficacy and safety of the inoculants, and the relationship of the trial test population to the total target population. We have limited the focus so far to the safety and efficacy issues since these constituted the core of what was presented to the FDA for EUA approval. We have not focused on the trials from an early warning indicator perspective.

We will address summarily the science/early warning indicator issues associated with the Pfizer trials, and how the neglect of these issues has translated into disastrous consequences during the mass inoculation rollout. Standard practice for determining and understanding the impact of new technology (such as mRNA "vaccines") on a system involves measuring the state and flux variables of the system before the new technology intervention, measuring the state and flux variables of the system after the new technology intervention, and identifying the types and magnitudes of changes in the state and flux variables attributable to the intervention. This would be in addition to evaluating performance metrics before and after the intervention.

In Pfizer's proposed clinical trials for the mRNA "vaccine" (Study to Describe the Safety, Tolerability, Immunogenicity, and Efficacy of RNA Vaccine Candidates Against COVID-19 in Healthy Individuals - https://clinicaltrials.gov/ct2/show/NCT04368728), the focus was on determining 1) adverse events/symptoms, 2) SARS-CoV-2 serum neutralizing antibody levels, 3) SARS-CoV-2 anti-S1 binding antibody levels and anti-RBD binding antibody levels, and 4) effectiveness. These metrics are all related to safety at the symptom level and performance.

However, symptoms/diseases are typically end points of processes that can take months, years, or decades to surface. During that symptom/disease development period, many biomarker early warning indicators tend to exhibit increasing abnormalities that reflect an increasing predisposition to the eventual symptom/disease. Thus, serious symptoms/diseases that ordinarily take long periods to develop would be expected to be rare events if they occurred shortly following an inoculation. If the clinical trials that were performed by Pfizer and Moderna were designed to focus on efficacy and *only adverse effects at the symptom level of description* as an indicator of safety, the trial results would be limited to the identification of rare events, and the trial results would potentially under-estimate the actual pre-symptom level damage from the inoculations.

Credible safety science applied to this experiment would have required a much more expansive approach to determining effects on a wide variety of state and flux metrics that could serve as early warning indicators of potentially serious symptoms/disease, and might occur with much higher frequencies at this early stage than the rare serious symptoms. The only mention of these other metrics in the above proposal is in the Phase I trial description: "Percentage of Phase 1 participants with abnormal haematology and chemistry laboratory values", to be generated seven days after dose 1 and dose 2.

A paper published in NEJM in December 2020 [34] summarized the Phase 1 results. The focus was on local and systemic adverse events and efficacy metrics (antibody responses). The only metrics other than these reported were transiently decreased lymphocyte counts.

We view this level of reporting as poor safety science for the following reasons. Before the clinical trials had started, many published articles were reporting serious effects associated with the presence of the SARS-CoV-2 virus such as hyperinflammation, hypercoagulation, hypoxia, etc. SARS-CoV-2 includes the S1 Subunit (spike protein), and it was not known how much of the damage was associated with the spike protein component of SARS-CoV-2. A credible high-quality safety science experiment would have required state measurements of specific biomarkers associated with each of these abnormal general biomarkers before and after the inoculations, such as d-dimers for evidence of enhanced coagulation/clotting; CRP for evidence of enhanced inflammation; troponins for evidence of cardiac damage; occludin and claudin for evidence of enhanced barrier permeability; blood oxygen levels for evidence of enhanced hypoxia; amyloid-beta and phosphorylated tau for evidence of increased predisposition to Alzheimer's disease; Serum HMGB1, CXCL13, Dickkopf-1 for evidence of an increased disposition to autoimmune disease, etc. A credible high-quality safety science experiment would have required flux measurements of products resulting from the mRNA interactions, from the LNP shell interactions, from dormant viruses that might have been stimulated by the mRNA-generated spike protein, etc., emitted through the sweat glands, faeces, saliva, exhalation, etc.

Most importantly, these types of measurements would have shown changes in the host that did not reach the symptom level of expression but raised the general level of host abnormality that could predispose the host to a higher probability of serious symptoms and diseases at some point in the future. Instead, in the absence of high-quality safety science reflected in these experiments, all that could be determined were short-term adverse effects and deaths. This focus on symptoms masked the true costs of the mRNA intervention, which would probably include much larger numbers of people whose health could have been degraded by the intervention as evidenced by increased abnormal values of these biomarkers. For example, the trials and VAERS reported clots that resulted in serious symptoms and deaths but gave no indication of the enhanced predisposition to forming serious clots in the future with a higher base of micro-clots formed because of the mRNA intervention. The latter is particularly relevant to children, who have a long future that could be seriously affected by having an increased predisposition to multiple clot-based (and other) serious diseases resulting from these inoculations.

3. Mass inoculation

3.1. Adverse events reported for adults

This section describes the adverse effects that followed COVID-19 mass inoculation in the USA. The main source of adverse effects data used was VAERS. Because VAERS is used to estimate adverse event information by many other countries as well, a short overview of VAERS and its intrinsic problems is summarized in Appendix 1.

The period in the present study covered by the reported inoculations is mid-December 2020 to the end of May 2021. The population inoculated during this period is mainly adults. Child inoculations did not begin until mid-May. Because the different age groups were inoculated starting at different times based on priority, the elapsed times after inoculation will be different, and any adverse event comparisons across age groups will require some type of elapsed post-inoculation time normalization.

We examined VAERS-reported deaths by age group, normalized to:

1) the number of inoculations given

2) the period within seven days after inoculation.

This allows a credible comparison of very short-term adverse effects post-inoculation for all age groups. During this period, which is eight days post-inoculation (where day zero is the day of inoculation), ˜sixty percent of all post-inoculation deaths are reported in VAERS.

Fig. 2 below shows the results circa late May 2021 [3]. The age band ranges are different from those in Fig. 1 because the CDC provides inoculation after-effect age bands differently from COVID-19 death age bands. In general, the inoculation deaths by age per inoculant roughly parallel the COVID-19 deaths by age per capita (the curve structures are very similar), with one exception: the 0–17 demographic. In the normalized COVID-19 death graph (Fig. 1), the deaths per capita in the 0–17 demographic are negligible, while in the normalized inoculant death graphs (Fig. 2) the normalized deaths are small, but not negligible. The members of the 65+ demographic, where the bulk of deaths are occurring in Fig. 1, Fig. 2, have been receiving inoculations for ˜five months, whereas the members of the youngest demographic have been receiving inoculations only for a few weeks. More time needs to pass before more definitive conclusions can be drawn about the youngest demographic, and how its members are impacted adversely following the inoculations.

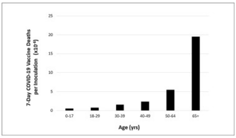

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Fig. 2. Post-inoculation deaths per dose of inoculant. 7-day COVID-19 vaccine deaths per inoculation by age in the United States (as of 5/28/2021). Data shown includes the total number of all deaths up to 7 days after receiving the vaccine for both those administered 1 dose and the complete series of doses by age in the United States as of 5/28/2021 reported in VAERS (updated on 5/28/2021). COVID-19 Vaccinations (Inoculations) based on CDC data provided by ISSInfo up thru 5/28/2021. Data obtained from https://data.cdc.gov/Vaccinations/COVID-19-Vaccination-Demographics-in-the-United-St/km4m-vcsb on 6/10/2021. COVID-19 Vaccinations Deaths based on CDC WONDER VAERS Database as of 5/28/2021, obtained from https://wonder.cdc.gov/controller/datarequest/D8;jsessionid=4B5522C8D1DA68F1A364646B0DA5 on 6/9/2021.

The high death rates from both COVID-19 and the inoculations in the 65+ demographic should not be surprising. In both cases, the immune system is challenged, and in both cases, a dysfunctional immune system characteristic of many elderly people with multiple comorbidities cannot respond adequately to the challenge.

3.1.1. Specific short-term adverse events reported in VAERS

The most comprehensive single evaluation of VAERS-reported adverse events (mainly for adult recipients of the COVID-19 “vaccines”) we have seen is a non-peer-reviewed collection of possible side effects by Dr. Ray Sahelian [35]. We recommend reading this short data-rich summary of the broad types of events reported already, in the context that these events are very short-term. Dr. Sahelian identifies five mechanisms he believes are responsible for most of these events, with research potentially uncovering other mechanisms. These five mechanisms include:

1. “An overreacting inflammatory response is known as systemic inflammatory response syndrome (SIRS). This SIRS reaction, perhaps a cytokine storm, can range from very mild to very severe. It can begin the very first day of the shot or begin days or weeks later as a delayed reaction.”
2. “Interaction of the spike proteins with ACE2 receptors on cell membranes. Such cells are found widely in the body including the skin, lungs, blood vessels, heart, mouth, gastrointestinal tract, kidneys, and brain.”
3. “Interaction of spike proteins with platelets and/or endothelial cells that line the inside of blood vessels. This can lead to clotting or bleeding (low number of circulating platelets in the bloodstream). Some of the clots, even if tiny, cause certain neurological symptoms if the blood supply to nerves is compromised.”
4. “Immediate or delayed release of histamine from mast cells and basophils (mast cell activation syndrome, MCAS).”
5. 5. “Swelling of lymph nodes in various areas of the body could interfere with blood flow, put pressure on nerves causing pain, or compromise their proper function.”

These reactions can be classified as Hyperinflammation, Hypercoagulation, Allergy, and Neurological, and can contribute to many symptoms and diseases, as VAERS is showing.

An excellent review of acute and potential long-term pathologies resulting from the COVID-19 inoculations [36] showed potential relationships to blood disorders, neurodegenerative diseases and autoimmune diseases. This review discussed the relevance of prion-protein-related amino acid sequences within the spike protein.

3.1.2. Potential mid- and long-term events and serious illnesses for

adults and children from past vaccines

A detailed description of potential mid- and long-term events and serious illnesses for adults and children from past vaccines is presented in Appendix C. Most of these events and illnesses are not predictable, and most, if not all, would be possible for the COVID-19 inoculations in the mid- and long-term for adults and children.

3.1.3. Potential short-, mid-, and long-term risks of mass COVID-19 inoculation for children

3.1.3.1. Intrinsic inoculant toxicity

Children are unique relative to COVID-19. They have negligible risks of serious effects from the disease, as shown in Fig. 1. Given that the COVID-19 inoculants were only tested for a few months, and mid-or long-term adverse effects are unknown, any mid- or long-term adverse events that emerge could impact children adversely for decades.

We believe that mid-or long-term adverse effects are possible based on the recent emergence of evidence that would support the probability of mid-and long-term adverse effects from the COVID-19 inoculants, such as:

1) The spike protein itself can be a toxin/pathogenic protein:

2) S protein alone can damage vascular endothelial cells (ECs) by downregulating ACE2 and consequently inhibiting mitochondrial function [37].

3) it is concluded that ACE2 and endothelial damage is a central part of SARS-CoV2 pathology and may be induced by the spike protein alone [38].

4) the spike protein of SARS-CoV-1 (without the rest of the virus) reduces ACE2 expression, increases angiotensin II levels, exacerbates lung injury, and triggers cell signaling events that may promote pulmonary vascular remodeling and Pulmonary Arterial Hypertension (PAH) as well as possibly other cardiovascular complications [39].

5) the recombinant S protein alone elicits functional alterations in cardiac vascular pericytes (PCs) [40]. This was documented as:

6) increased migration

7) reduced ability to support EC network formation on Matrigel

8) secretion of pro-inflammatory molecules typically involved in the cytokine storm

9) production of pro-apoptotic factors responsible for EC death. Furthermore, the S protein stimulates the phosphorylation/activation of the extracellular signal-regulated kinase 1/2 (ERK1/2) through the CD147 receptor, but not ACE2, in cardiac PCs, the S protein may elicit vascular cell dysfunction, potentially amplifying, or perpetuating, the damage caused by the whole coronavirus [40].

10) "even in the absence of the angiotensin-converting enzyme 2 receptors, the S1 subunit from SARS-CoV-2 spike protein binding to neutral phospholipid membranes leads to their mechanical destabilization and permeabilization. A similar cytotoxic effect of the protein was seen in human lung epithelial cells." [125].

11) The LNP layer encapsulating the mRNA of the inoculant is highly inflammatory in both intradermal and intranasal inoculation [41] and "Polyethylene glycol (PEG) is a cause of anaphylaxis to the Pfizer/BioNTech mRNA COVID-19 vaccine" [42]. "Humans are likely developing PEG antibodies because of exposure to everyday products containing PEG. Therefore, some of the immediate allergic responses observed with the first shot of mRNA-LNP vaccines might be related to pre-existing PEG antibodies. Since these vaccines often require a booster shot, anti-PEG antibody formation is expected after the first shot. Thus, the allergic events are likely to increase upon re-vaccination" [43].

    There is also the possibility that the components of the LNP shell could induce the ASIA Syndrome (autoimmune/inflammatory syndrome induced by adjuvants), as shown by studies on post-inoculation thyroid hyperactivity [44] and post-inoculation subacute thyroiditis [45].

12 The spike protein has been found in the plasma of post-inoculation individuals, implying that it could circulate to, and impact adversely, any part of the body [46].

13. The spike protein of SARS-CoV-2 crosses the blood-brain barrier in mice [47], and “the SARS-CoV-2 spike proteins trigger a pro-inflammatory response on brain endothelial cells that may contribute to an altered state of BBB function” [48].

14. The spike proteins manufactured in vivo by the present COVID-19 inoculations could potentially "precipitate the onset of autoimmunity in susceptible subgroups, and potentially exacerbate autoimmunity in subjects that have pre-existing autoimmune diseases", based on the finding that anti-SARS-CoV-2 protein antibodies cross-reacted with 28 of 55 diverse human tissue antigens [49].

15. “The biodistribution of ChaAdOx1 [Astra Zeneca’s recombinant adenovirus vaccine candidate against SARS-CoV-2] in mice confirmed the delivery of vaccine into the brain tissues [50]. The vaccine may therefore spur the brain cells to produce CoViD spike proteins that may lead to an immune response against brain cells, or it may spark a spike protein-induced thrombosis. This may explain the peculiar incidences of the fatal cerebral venous sinus thrombosis (CVST) observed with viral vector-based CoViD-19 vaccines” [51,52].

    A complementary perspective to explain adenovirus-based vaccine-induced thrombocytopenia is that “transcription of wildtype and codon-optimized Spike open reading frames enables alternative splice events that lead to C-terminal truncated, soluble Spike protein variants. These soluble Spike variants may initiate severe side effects when binding to ACE2-expressing endothelial cells in blood vessels.” [100].

16. A Pfizer Confidential study performed in Japan showed that "modRNA encoding luciferase formulated in LNP comparable to BNT162b2″ injected intramuscularly concentrated in many organs/tissues in addition to the injection site [53]. The main organs/sites identified were adrenal glands, liver, spleen, bone marrow, and ovaries. While damage to any of these organs/sites could be serious (if real for humans), adverse effects on the ovaries could be potentially catastrophic for women of childbearing or pre-childbearing age.

The main objective of credible biodistribution studies (of inoculants for eventual human use) is to identify the spatio-temporal distribution of the actual inoculant in humans; i.e., how much of the final desired product (in this case, expressed protein antigen/spike protein) is produced in different human tissues and organs as a function of time. That’s not what was reported in the Pfizer Confidential study.

Rats were used for the in vivo studies; the relationship of their biodistribution to that of humans is unclear. They were injected in different locations (hindpaw/intramuscular); the relationship to human injections in the deltoid muscle is unclear. They were injected with "modRNA encoding luciferase formulated in LNP comparable to BNT162b2″; it is unclear why they weren’t injected with BNT162b2, it is unclear why spike protein expression wasn’t evaluated rather than LNP concentration, and it is unclear how well the biodistribution from the actual inoculant used in the experiments compares to the biodistribution from BNT162b2.

They were injected once per rat. Given that a second injection would not be in the same exact location as the first, and that the circulatory system might have changed due to clotting effects from the first injection and other potential vascular complications, it is unclear how the biodistribution change with the second injection would compare with the first. If a booster injection is given to counter variants, it is unclear how its biodistribution would be altered as a consequence of the preceding two injections.

Clotting will occur with the highest probability where the blood flow is reduced (and more time is available for LNP-endothelial cell interaction). It is unclear whether the clotting process would show *positive feedback* behaviour where the initial inoculation constricts the flow in low-velocity regions even further by enhanced clotting, and subsequent inoculations further amplify this reduced flow-enhanced clotting cycle.

The rats were injected under pristine conditions; how that compares with humans, who have been, are being, and will continue to be exposed to multiple toxic substances in combination, is open to question. We know these combinations can act synergistically to adversely impact myriad organs and tissues throughout the body [23]. We don’t know how these toxic exposures in humans affect the permeability of the blood/tissue barriers, and especially the ability of the injected material to diffuse into the bloodstream (and also the ability of the manufactured spike proteins to diffuse from the bloodstream into the surrounding tissue).

Higher-level primates should have been used for these short-term experiments, to obtain a more realistic picture of the biodistribution of inoculant in human organs and tissues. In other words, these laboratory experiments may be just the tip of the iceberg of estimating the amount of inoculant that concentrates in critical organs and tissues of human beings.

The many studies referenced above indicate collectively that the mRNA-based COVID-19 inoculations (the most prolific inoculations used in the USA for COVID-19 so far) consist of (at least) two major toxins: the instructions for the spike protein (mRNA) and the mRNA-encapsulating synthetic fat LNP. The vaccine is injected into the deltoid muscle, at which time it contributes to inflammation at the injection site due in part to the LNP and potentially to anaphylaxis from the LNP PEG-2000 component. Some of the injected material stays at the injection site, where it combines with cells through endocytosis to express spike protein on the cell surface, stimulating the adaptive immune system to eventually produce antibodies to the spike protein [54].

The remainder of the injected material enters the lymphatic system and the bloodstream, and is distributed to tissues and organs throughout the body: e.g., "Drugs administered by the intramuscular (IM) route are deposited into vascular muscle tissue, which allows for rapid absorption into the circulation" [55]. The basis of this process is that the bulky muscles have good vascularity, and therefore the injected drug quickly reaches the systemic circulation and thereafter into the specific region of action, bypassing the first-pass metabolism [56]. The widespread distribution is greatly enhanced by the LNP PEG-2000 coating as follows: building from the success of PEGylating proteins to improve systemic circulation time and decrease immunogenicity [57]. PEG coatings on nanoparticles shield the surface from aggregation, opsonization, and phagocytosis, prolonging systemic circulation time. [57]. PEG coatings on nanoparticles have also been utilized for overcoming various biological barriers to efficient drug and gene delivery associated with other modes of administration. [57]

In the bloodstream, one possible outcome is that the LNPs coalesce with the endothelial cells on the inner lining of the blood vessels and transfer the mRNA to the cells through endocytosis. The endothelial cells would then express the spike protein on their surface. Platelets flowing by the spike protein express ACE2 receptors on their surface; therefore, one possible outcome would be activation of the platelets by the spike protein and initiation of clotting. Another possible outcome would be the modified endothelial cells being recognized by innate immune system cells as foreign. These immune killer cells would then destroy parts of the endothelium and weaken the blood-organ barriers. The LNPs would inflame the endothelium as well, both increasing barrier permeability and increasing the blood vessel diameter. This weakening of the blood-organ barriers would be superimposed on any inflammation due to the myriad toxic contributing factors operable [4]. The newly-formed cells with spike proteins would penetrate the blood-organ barriers and bind to tissue with expressed ACE2 receptors. Any LNPs that did not coalesce with the endothelial cells, but remained intact, could also pass through the permeable blood-organ barrier, and coalesce directly with the organ cells. This could lead to an attack by innate immune system cells, and be a precursor to autoimmunity [4].

In the preceding discussion of the Pfizer biodistribution studies, the issue of multiple inoculations on changes in biodistribution was raised. Similarly, the alteration of effects as described above by multiple inoculations must be considered. Each inoculation will have positive aspects and negative aspects. The positive aspects are the formation of antibodies in the muscle cells and lymphatic system. The negative aspects include, but are not limited to, the potential clotting effects and permeability increases for that fraction of the inoculant that enters the bloodstream. The first inoculant dose can be viewed as priming the immune system. The immune response will be relatively modest. The second inoculant dose can be expected to elicit a more vigorous immune response. This will enhance the desired antibody production in the muscle cells and lymphatic system, but may also enhance the immune response to both the blood vessel-lining endothelial cells displaying the spike protein and the platelets, causing more severe damage. If a booster(s) inoculation is also required, this may further enhance both the positive and negative immune responses resulting from the second inoculation. While the positive effects are reversible (antibody levels decrease with time), adverse effects may be cumulative and irreversible, and therefore injury and death rates may increase with every additional inoculation [58].

These effects can occur throughout the body in the short term, as we are seeing with the VAERS results. They can occur in the mid- and long-term as well, due to the time required for destructive processes to have full effect and the administration of further inoculations. For example, micro-clots resulting from the inoculation that were insufficient to cause observable symptoms could in effect raise the baseline for thrombotic disease [92]. Lifestyle activities that contribute to enhanced blood clotting would have less distance to travel to produce observable symptoms, and thus the serious effects of clotting would have been accelerated [59,60]. As an example: the risk of venous thrombosis is approximately 2- to 4-fold increased after air travel [61]. How much this rate would increase after the inoculations, where microthrombi have formed in some recipients, is unknown. These potential baseline-raising effects could impact the interpretation of the VAERS results, as we show at the end of Appendix 1.

3.1.3.2. Adverse inoculant effects on children

What are the potential mid- and long-term adverse health effects from the COVID-19 inoculation on children specifically, taking into account that they will be exposed not only to the spike protein component of the SARS-CoV-2 virus but also to the toxic LNP encapsulating-shell? This toxic combination will have bypassed many defensive safeguards (typically provided by the innate immune system) through direct injection [62]. As we have shown, the main reasons why we believe the spike protein could be harmful to children even though they don't seem to get sick from exposure to SARS-CoV-2 are 1) the bypassing of the innate immune system by inoculation, 2) the larger volume of spike protein that enters the bloodstream, and 3) the additional toxic effects of the encapsulating LNP layer.

3.1.3.2.1. Potential mid-term adverse health effects

Examination of the myriad post-COVID-19 inoculation symptoms/biomarker changes for the 0–17 age demographic reported to VAERS circa mid-June 2021 provides some indication of very early damage [84]. Main regions/systems affected adversely (VAERS symptoms/biomarkers shown in parentheses) include:

- Cardiovascular (blood creatine phosphokinase increased, cardiac imaging procedure abnormal, echocardiogram abnormal, electrocardiogram abnormal, heart rate increased, myocarditis, palpitations, pericarditis, tachycardia, troponin I increased, troponin increased, fibrin D-Dimer increased, platelet count decreased, blood pressure increased, bradycardia, brain natriuretic peptide increased, ejection fraction decreased, migraine)
- Gastrointestinal (abdominal pain, diarrhoea, vomiting, alanine aminotransferase increased, aspartate aminotransferase increased.)
- Neural (gait disturbance, mobility decreased, muscle spasms, muscle twitching, seizure, tremor, Bell's Palsy, dyskinesia)
- Immune (C-Reactive Protein increased, red blood cell sedimentation rate increased, white blood cell counts increased, inflammation, anaphylactic reaction, pruritis, rash, lymphadenopathy)
- Endocrine (heavy menstrual bleeding, menstrual disorder)

In addition, there were large numbers of different vision and breathing problems reported.

All the major systems of the body are impacted, and many of the major organs as well. Given the lag times in entering data into VAERS and the fact that inoculations of children started fairly recently, we would expect the emphasis to be immediate symptomatic and biomarker reactions. More time is required for organ and system damage to develop and emerge. Cardiovascular problems dominate, as our model for spike protein/LNP circulation and damage predicts, and it is unknown how reversible such problems are. Many of the VAERS symptoms listed above were also found in COVID-19 adult patients [64].

Consider the example of Multisystem Inflammatory Syndrome in Children (MIS-C). It has emerged in VAERS with modest frequency so far, and it also occurred about a month after COVID-19 infection [65]. In both cases, the presence of the spike protein was a common feature. Many of its characteristic symptoms are those listed above from VAERS. MIS-C has similarities with known disease entities like Kawasaki Disease (KD), toxic shock syndrome (TSS) and macrophage activation syndrome (MAS)/secondary hemophagocytic lymphohistiocytosis (HLH) [66]. One presentation of MIS-C is in adolescents with a high disease burden as evidenced by more organ systems involved, almost universally including cardiac and gastrointestinal systems, and with a higher incidence of shock, lymphopenia, and elevated cardiac biomarkers indicating myocarditis [67]. Since the first reports of children developing MIS-C, it was evident that others presented with some of the classic symptoms of the well-recognized childhood illness KD [68]. Further, despite KD being ordinarily incredibly rare in adults, patients with MIS-A have also been reported with KD-like features. [68] Thus, an examination of the adverse effects from COVID-19 as evidenced through these diseases might shed some light on what can be expected further down the line from the inoculations.

The following section addresses Kawasaki disease (KD) and Multisystem Inflammatory Syndrome in Children (MIS-C) [65].

KD is an acute vasculitis and inflammation that predominantly affects the coronary arteries and can cause coronary artery aneurysms. Other KD manifestations include systemic inflammation of arteries, organs, and tissues, with consequent hepatitis and abdominal pain; lung interstitial pneumonitis, aseptic meningitis due to brain membrane inflammations; myocarditis, pericarditis, and valvulitis; urinary tract pyuria, pancreatitis; and lymph-node enlargement [69]. In general, although almost all children fully recover, some of them later develop coronary artery dilation or aneurysm [70]. Etiologically and pathologically, numerous studies indicate that KD is triggered by an abnormal autoimmune response caused by an infection [71]. The infection hypothesis is supported by epidemiology data showing that an infectious disease is involved at least as a starting point. Previously proposed infectious agents include Herpesviridae, retroviruses, Parvovirus B19, bocavirus, and bacterial infections such as staphylococci, streptococci, Bartonella, and Yersinia infections [72].

SARS-CoV-2 adds to these infectious agents by eliciting autoantibodies likely via molecular mimicry and cross-reactivity with autoantigens [72,73].

Then, the formation of antigen–antibody immune complexes can lead to KD symptoms via activation of the receptors of mast cells, neutrophils, and macrophages with consequent release of pro-inflammatory cytokines and increase of blood vessel permeability; activation of the complement system, stimulation of neutrophils and macrophages to secrete proteases and more proinflammatory cytokines [74], thus merging into the “cytokine storm” that characterizes MIS-C [75]. Indeed, features of KD are raised levels of Interleukin (IL)-6, IL-8, IL-15, and IL-17, with the cytokine level predicting coronary aneurysm formation in KD patients [76,77]

3.1.3.2.2. Potential long-term adverse health effects

In the long-term, SARS-CoV-2-induced KD vasculitis can lead to severe pathologies. Vasculitis has a predilection for coronary arteries with a high complication rate across the lifespan for those with medium to large coronary artery aneurysms [78]. The cytokine-induced inflammation produces endothelial dysfunction and damage to the vascular wall, leading to aneurysmal dilatation. Successively, vascular remodeling can also occur, but this does not imply resolution of the disease or reduction of risk for future complications. A rigorous follow-up to detect progressive stenosis, thrombosis and luminal occlusion that may lead to myocardial ischemia and infarction becomes mandatory [78]. Of equal importance, among other long-term outcomes, children with KD may have increased risks not only for ischemic heart disease, but also for autoimmune disorders, cancer as well as an increased all-cause mortality [71].

Additional questions regarding mass inoculation of children and adolescents include:

a) Do children, being asymptomatic carriers of SARS-CoV-2, transmit the virus?

b) Do recently vaccinated people, infected with SARS-CoV-2, transmit the virus?

There is evidence of children transmitting SARS-CoV-2 in community settings, but the existing literature is heterogeneous with regards to the relative rate at which they do so compared to adults [79].

Studies from South Korea and Thailand found a very limited number of secondary cases [80,81]. On the contrary, a large contact tracing study from India concluded that the highest probability of transmission was between case-contact pairs of similar age and that this pattern of enhanced transmission risk was highest among children 0–4 years of age as well as adults 65 years of age and older [80]

With regard to the second question, it was shown that household members of healthcare workers inoculated with a single dose of either Pfizer or Astra

Zeneca COVID-19 inoculant were at significantly reduced risk of PCR-confirmed SARS-CoV-2 infection but at non-statistically significant reduced risk of hospitalization, compared to household members of uninoculated healthcare workers, fourteen days after inoculation [82]. This finding again underlines the association of severe disease to the characteristics of the infected person and not directly to the transmission, implying that the elderly should be inoculated and not the children.

3.2. Novel best-case scenario cost-benefit analysis of COVID-19 inoculations for most vulnerable

Traditional cost-benefit analyses are typically financial tools used to estimate the potential value of a proposed project. They involve generating cost streams over time, benefit streams over time, and then comparing the net present value of these two streams (including risk) to see whether the risk-adjusted discounted benefits outweigh the risk-adjusted discounted costs. Appendix D presents a detailed non-traditional *best-case scenario* pseudo-cost-benefit analysis of inoculating people in the 65+ demographic in the USA. In this incarnation of a cost-benefit analysis, the costs are the number of deaths resulting from the inoculations, and the benefits are the lives saved by the inoculations. The time range used was from December 2019 to end-of-May 2021. No discounting was done; an inoculation-based death occurring immediately post-inoculation was given the same importance/weighting as an inoculation-based death months after inoculation.

Why was this non-traditional approach selected for a cost-benefit analysis? In a traditional non-financial cost-benefit analysis relative to inoculations, the adverse events prevented by the inoculations would be compared with the adverse events resulting from the inoculations. Presently, in the USA, definitions, test criteria, and reporting incentives for COVID-19 and its inoculants have shifted over time, and we believe a standard approach could not be performed credibly. Appendix Da presents some of the problems with the COVID-19 diagnostic criteria on which the above statements are based.

In contrast to the pandemic buildup phase, where many who died *with* COVID-19 were assumed to have died *from* COVID-19 by the medical community and the CDC, the post-inoculation deaths reported in VAERS are assumed by the CDC to be mostly from causes other than the inoculations. We wanted to use a modified cost-benefit analysis that would have less dependence on arbitrary criteria and subjective judgments.

The approach selected can be viewed as a *best-case scenario* pseudo-cost-benefit analysis. We assume the inoculations prevent ***all*** the deaths ***truly*** attributable to COVID-19 (these are the total deaths attributed to COVID-19 officially minus 1) the number of false positives resulting from the PCR tests run at very high amplification cycles and 2) the number of deaths that could have been attributed to one of the many comorbidities that were typical of those who succumbed, as shown in our results section) over the period December 2019 to end-of-May 2021, and relate that number to the deaths ***truly*** attributable to the inoculation (from January 2021 to end-of-May 2021) based on our computations in the results section. The results show ***conservatively*** that there are five times the number of deaths ***truly*** attributable to each inoculation vs those ***truly*** attributable to COVID-19 in the 65+ demographic. As age decreases, and the risk for COVID-19 decreases, the cost-benefit increases. Thus, if the best-case scenario looks ***poor*** for benefits from the inoculations, any realistic scenario will look ***very poor***. For children the chances of death from COVID-19 are negligible, but the chances of serious damage over their lifetime from the toxic inoculations are not negligible.

4. Discussion

Two issues arise from these results.

First, where is the data justifying inoculation for children, much less most people under forty? It's not found on Fig. 1, where the most vulnerable are almost exclusively the elderly with many comorbidities [83]. Yet, in the USA, Pfizer has been approved to inoculate children 12–17, and the goal is to accomplish this by the start of the school year in the Fall. As stated previously, there are plans to inoculate children as young as six months starting before the end of 2021.

What is the rush for a group at essentially zero risks? Given that the inoculations were tested only for a few months, only very short-term adverse effects could be obtained. It is questionable how well even these short-term effects obtained from the clinical trials reflect the short-term effects from the initial mass inoculation results reported in VAERS.

Fig. 1, Fig. 2 reflect only these very short-term results. A number of researchers have suggested the possibility of severe longer-term autoimmune, Antibody-Dependent Enhancement, neurological, and other potentially serious effects, with lag periods ranging from months to years. If such effects do turn out to be real, the children are the ones who will have to bear the brunt of the suffering. There appear to be no benefits for the children and young adults from the inoculations and only Costs!

The second issue is why the deaths shown on Fig. 2 were not predicted by the clinical trials. We examined the Pfizer trial results (based on a few months of testing) and did not see how (potentially) hundreds of thousands of deaths could have been predicted from the trials' mortality results. Why this gap?

As we showed in the clinical trials section, 17.4 % of the Pfizer sample members were over 65, and 4.4 % were over 75. When the later phases of the trials started in late July 2020, the managers knew the COVID-19 age demographics affected from the July 2020 analog of Fig. 1. Rather than sampling from the age region most affected, they sampled mainly from the age region least affected! And even in the very limited sampling from the oldest groups, it is unclear whether they selected from those with the most serious comorbidities. Our impression is that the sickest were excluded from the trials, but were first in line for the inoculants.

It is becoming clear that the central ingredient of the injection, the recipe for the spike protein, will produce a product that can have three effects. Two of the three occur with the production of antibodies to the spike protein. These antibodies could allegedly offer protection against the virus (although with all the "breakthrough" cases reported, that is questionable), or could suppress serious symptoms to some extent. They could also cross-react with human tissue antigen, leading to potential autoimmune effects. The third occurs when the injected material enters the bloodstream and circulates widely, which is enabled by the highly vascular injection site and the use of the PEG-2000 coating.

This allows spike protein to be manufactured/expressed in endothelial cells at any location in the body, both activating platelets to cause clotting and causing vascular damage. It is difficult to believe this effect is unknown to the manufacturer, and in any case, has been demonstrated in myriad locations in the body using VAERS data. There appears to be modest benefit from the inoculations to the elderly population most at risk, no benefit to the younger population not at risk, and much potential for harm from the inoculations to both populations. It is unclear why this mass inoculation for all groups is being done, being allowed, and being promoted.

5. Overall conclusions

The people with myriad comorbidities in the age range where most deaths with COVID-19 occurred were in very poor health. Their deaths did not seem to increase all-cause mortality as shown in several studies. If they hadn't died with COVID-19, they probably would have died from the flu or many of the other comorbidities they had. We can't say for sure that many/most died from COVID-19 because of: 1) how the PCR tests were manipulated to give copious false positives and 2) how deaths were arbitrarily attributed to COVID-19 in the presence of myriad comorbidities.

The graphs presented in this paper indicate that the frail injection recipients receive minimal benefit from the inoculation. Their basic problem is a dysfunctional immune system, resulting in part or in whole from a lifetime of toxic exposures and toxic behaviors. They are susceptible to either the wild virus triggering the dysfunctional immune system into over-reacting or under-reacting, leading to poor outcomes or the injection doing the same.

This can be illustrated by the following analogy. A person stands in a bare metal enclosure. What happens when the person lights a match and drops it on the floor depends on what is on the floor. If the floor remains bare metal, the match burns for a few seconds until extinguished. If there is a sheet of paper on the floor under the match, the match and the paper will burn for a short time until both are extinguished. If, however, the floor is covered with ammonium nitrate and similar combustible/explosive materials, a major explosion will result! For COVID-19, the wild virus is the match. The combustible materials are the toxic exposures and toxic behaviors. If there are no biomarker 'footprints' from toxic exposures and toxic behaviors, nothing happens. If there are significant biomarker 'footprints' from toxic exposures and toxic behaviors, bad outcomes result.

Adequate safety testing of the COVID-19 inoculations would have provided a distribution of the outcomes to be expected from 'lighting the match'. Since adequate testing was not performed, we have no idea how many combustible materials are on the floor, and what the expected outcomes will be from 'lighting the match'.

The injection goes two steps further than the wild virus because 1) it contains the instructions for making the spike protein, which several experiments are showing can cause vascular and other forms of damage, and 2) it bypasses many front-line defenses of the innate immune system to enter the bloodstream directly in part. Unlike the virus example, the injection ensures there will always be some combustible materials on the floor, even if there are no other toxic exposures or behaviors. In other words, the spike protein and the surrounding LNP are toxins with the potential to cause myriad short-, mid-, and long-term adverse health effects even in the absence of other contributing factors! Where and when these effects occur will depend on the biodistribution of the injected material. Pfizer's own biodistribution studies have shown the injected material can be found in myriad critical organs throughout the body, leading to the possibility of multi-organ failure. And these studies were from a single injection. Multiple injections and booster shots may have cumulative effects on organ distributions of inoculant!

The COVID-19 reported deaths are people who died **with** COVID-19, not necessarily **from** COVID-19. Likewise, the VAERS deaths are people who have died **following** inoculation, not necessarily **from** inoculation.

As stated before, CDC showed that 94 % of the reported deaths had multiple comorbidities, thereby reducing the CDC's numbers attributed strictly to COVID-19 to about 35,000 for all age groups. Given the number of high false positives from the high amplification cycle PCR tests, and the willingness of healthcare professionals to attribute death to COVID-19 in the absence of tests or sometimes even with negative PCR tests, this 35,000 number is probably highly inflated as well.

On the latter issue, both Virginia Stoner [85] and Jessica Rose [86] have shown independently that the deaths **following** inoculation are not coincidental and are **strongly related to** inoculation through strong clustering around the time of injection. Our independent analyses of the VAERS database reported in Appendix 1 confirmed these clustering findings.

Additionally, VAERS historically has under-reported adverse events by about two orders-of-magnitude, so COVID-19 inoculation deaths ***in the short-term*** could be in the hundreds of thousands for the USA for the period mid-December 2020 to the end of May 2021, potentially swamping the *real* COVID-

19 deaths. Finally, the VAERS deaths reported so far are for the very short term. We have no idea what the death numbers will be in the intermediate and long-term; the clinical trials did not test for those.

The clinical trials used a non-representative younger and healthier sample to get EUA for the injection. Following EUA, the mass inoculations were administered to the very sick (and first responders) initially, and many died quite rapidly. However, because the elderly who died following COVID-19 inoculation were very frail with multiple comorbidities, their deaths could easily be attributed to causes other than the injection (as should have been the case for COVID-19 deaths as well).

Now the objective is the inoculation of the total USA population. Since many of these potential serious adverse effects have built-in lag times of at least six months or more, we won't know what they are until most of the population has been inoculated, and corrective action may be too late.

All the authors contributed equally and approved the final version of the manuscript.

Author’s contribution

Kostoff RN contributed to this paper with conception, data analysis, and writing the manuscript; Calina D contributed to data analysis, writing the manuscript, and editing; Kanduc D participated in data analysis and writing the manuscript; Briggs MB participated in data analysis, results validation, and graphics development; Vlachoyiannopoulos P participated in writing the manuscript; Svistunov AA participated in editing and reviewing the manuscript; Tsatsakis A participated in editing and reviewing the manuscript; all the authors contributed equally and approved the final version of the manuscript.

Ethical approval

Not applicable.

Declaration of Competing Interest

The authors declare that they have no competing interests. Aristides Tsatsakis is the Editor-in-Chief for the journal but had no personal involvement in the reviewing process, or any influence in terms of adjudicating on the final decision, for this article.

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Appendix A

EXPECTED DEATHS IN 65+ DEMOGRAPHIC VS COVID-19 INOCULATION DEATHS

The goal of this appendix is to estimate the number of actual deaths from the COVID-19 inoculation based on the number of deaths following inoculation reported in VAERS [93,94,101]. The approach used will:

1) identify the number of deaths following COVID-19 inoculation that would have been **expected** without COVID-19 inoculation (i.e., pre-COVID-19 death statistics);
2) relate the VAERS **expected** death data to the actual number of deaths **expected** based on historical death statistics; and
3) apply this ratio to scale-up the deaths attributed to COVID-19 inoculation reported in VAERS to arrive at actual deaths attributable to COVID-19 inoculation.

For example, if ten deaths could be shown in VAERS to reflect expected pre-COVID-19 deaths, and the actual number of expected pre-COVID-19 deaths from historical data was 100, the scaling factor of deaths would be ten to translate VAERS-reported deaths to actual deaths. Then, the deaths reported in VAERS that can be attributed to the COVID-19 inoculation will be multiplied by the expected deaths scaling factor, ten, to arrive at the actual number of deaths resulting from the COVID-19 inoculation. Thus, if VAERS shows fifty deaths that can be attributed to the COVID-19 inoculation, then the actual number of deaths attributed to COVID-19 will be 500 with these assumptions [3].

The basis for our approach is the following statement from the USA Federal government: “Healthcare providers are required to report to VAERS the following adverse events after COVID-19 vaccination [33] and other adverse events if later revised by FDA" [96,102,103]. "Serious AEs regardless of causality.", including death [3,95].

If there had been full compliance with this requirement in VAERS, then the VAERS-reported deaths would have equaled the sum of

1) actual expected deaths (based on past statistics)

2) actual deaths over and above expected deaths that could be attributed to the COVID-19 inoculations.

Based on this requirement, we will generate a rough estimate (in the simplest form possible) of the number of deaths that would have occurred in the 65+ demographic if there had been no COVID-19 “pandemic”. Then, we will relate this number to the number of deaths reported to VAERS following COVID-19 inoculations in the 65+demographic. This would provide a “floor” for estimating the fraction of actual deaths reported to VAERS. This will be followed by parameterizing potential deaths attributable to the COVID-19 inoculations and displaying the effects on ratio of reported deaths to actual deaths. We will perform a global analysis and a local analysis, to see whether major or minor differences occur. The local analysis (Section A1-a2) may be somewhat easier to comprehend than the global analysis, but both come to similar conclusions.

A1-a Deaths Following COVID-19 Inoculations Reported to VAERS Compared to Expected Deaths

A1-a . Problems with VAERS

Before we discuss numbers of adverse events reported by VAERS, we need to identify potential shortcomings of, and problems with, VAERS, so these numbers of adverse events can be understood in their proper context. As stated previously, VAERS is a passive surveillance system managed jointly by the CDC and FDA, and historically has been shown to report about 1% of actual vaccine/inoculation adverse events (confirmed by the first principles analysis that follows in this appendix). There is no evidence that even the 1% reported have been selected randomly.

Some of this gross underreporting of adverse events reflects a major conflict-of-interest of CDC with respect to VAERS. CDC provides funding for administration of many vaccines, including the COVID-19 inoculations. Prior to COVID-19, the CDC provided about five billion dollars annually to the Vaccines for Children Program alone [102].

For COVID-19, the CDC has received many billions of dollars in supplemental funding for myriad activities, including vaccine distribution. It is difficult to separate out the CDC funding available for vaccine distribution from other CDC COVID-19 related activities, but one budget item (of many) should illustrate the magnitude of the effort: “Coronavirus Response and Relief Supplemental Appropriations Act, 2021 (P.L. 116–260): P.L. 116–260 provided $8.75 billion to CDC to plan, prepare for, promote, distribute, administer, monitor, and track coronavirus vaccines to ensure broad-based distribution, access, and vaccine coverage.” [3]. Low reporting rates of actual adverse events in VAERS should not be surprising, since the same organization that receives multi-billions of dollars in funding annually for promoting and administering vaccines also has responsibility for monitoring the safety of these products (whose liability has been waived).

In addition, the 1% reporting rates came from a thirty-day tracking study [22], and therefore are strictly applicable to *very near-term* adverse events. For mid-term and especially long-term events, the reporting rates would be much lower, since the links between inoculation and adverse events would be less obvious. That doesn’t mean these non-very-short-term adverse events don’t exist; it just means they haven’t been tracked. Absence of evidence is not evidence of absence. Thus, the VAERS numbers should be viewed as a very low “floor’ of the numbers and types of adverse events from COVID-19 inoculations that exist in the real-world.

A1-a2 Global analysis

We used 2019 death statistics from CDC to start the analysis. According to search results from CDC Wonder [104] obtained 11 June 2021, there were 2,117,332 deaths from all causes for people aged 65+ in the United States in 2019. Assuming uniformity throughout the year, there would have been

~882,000 deaths occurring the first five months of the year, and that number will be used as the expected deaths for the first five months of 2021. From the same source, the population estimate is ~54,000,000 for the 65+ age range. From CDC COVID-19 data tracker, the number of people 65+ vaccinated with at least one dose is ~44,000,000 [24]

For those who were inoculated somewhere in the time frame 1 January 2021 to 31 May 2021, the number who would have been expected to die in the period from inoculation to 31 May will be a function of the duration of this period. For example, if all 44,000,000 people had been fully inoculated on 1 January 2021, then the number expected to die post-inoculation from non-COVID-19 inoculation causes would be simply (44,000,000/54,000,000) x 882,000, or ~723,000 deaths. Conversely, if all 44,000,000 people had been fully inoculated on 31 May 2021, then the number expected to die post-inoculation from non-COVID-19 inoculation causes would be extremely small [24].

For an accurate estimation of the number expected to die post-inoculation from non-COVID-19 causes, one would need to integrate the time between inoculation and 31 May over the inoculation temporal distribution function. For present purposes, we will do a very rough approximation by modeling the inoculation distribution function as a delta function occurring at a mean temporal location. In other words, we compress all inoculations an individual receives into one, identify the mean temporal location from the actual inoculation distribution function, and compute the expected deaths based on the distance from 31 May to the temporal mean point.

From a graph of inoculation trends in the CDC data tracker [101] the distribution appears to be non-symmetrical pyramidal, rising to a peak in mid-April. This is slightly over the 2/3 point in the five-month range of interest. We will approximate the mean time point as 2/3 of the distance.

Table A1 displays the mean time normalized to the five-month study window vs potential deaths from COVID-19 inoculation (not expected from prior census data) normalized to the deaths expected from prior census data. Each cell represents the percent of deaths reported in VAERS following inoculation relative to total deaths (number of deaths expected from prior census data plus number of deaths following COVID-19 inoculation not contained in the expected death group). The model on which the table is based is as follows: there are two classes of deaths for the period following COVID-19 inoculation. One is the deaths expected from prior census data, and the other is deaths attributable mainly to COVID-19 inoculation. There would be potentially substantial overlap between the two in this age group (and perhaps other age groups as well). We assume that we can tag those individuals who would be expected to die based on prior census data. The remaining deaths attributable to COVID-19 inoculation not contained within the tagged group are classified as potential COVID deaths in Table A1.

Table A1. Expected deaths from non-COVID-19 causes for inoculees (Thousands).

Potential covid deaths/# non-covid expected	**Mean time location/five months**									
	0	**%REP**	**1/3**	**%REP**	**1/2**	**%REP**	**2/3**	**%REP**	**1**	**%REP**
0	723	0.5	482	0.74	362	0.98	242	1.47	4.77	75
.5	1085	0.33	723	0.5	543	0.66	363	0.98	7.14	50
1	1446	0.25	964	0.37	724	0.49	484	0.74	9.51	37

Consider the cell (2/3,0). The mean time is about mid-April 2021 and the only deaths occurring are those expected (some may have died because of the inoculation, but they were sufficiently ill that they would have died during that period without the inoculation). There were 723,000 expected deaths and ~3560 reported, yielding a ratio of deaths reported in VAERS to actual deaths of ½%.

Consider the cell (1/2,1). The mean time would have been about mid-March 2021 and the inoculation distribution would have resembled an isosceles triangle. The total deaths occurring are those expected and an equal number whose deaths were attributed to COVID-19 inoculation but did not overlap with those in the tagged expected group (there still could have been some/many in the latter group that may have died because of the inoculation, but they were sufficiently ill that they would have died during that period without the inoculation). There were 724,000 total deaths that occurred during that period and ~3560 reported, yielding a ratio of deaths reported in VAERS to actual deaths of ½%. [3]

So, according to Table A1, focusing on the parameter most closely reflecting the actual inoculation distribution (2/3), the reporting percentages of actual to total are about 1%. This mirrors the Harvard Pilgrim study results (referenced in our vaccine safety study) which were obtained through an entirely different empirical approach [4]. At least for deaths reporting, there appears to be an approximately two order of magnitude difference between actual and reported deaths in VAERS.

Table A1 used two parameters to examine a broad spectrum of possible results, the mean time and the number of deaths solely attributable to COVID-19 inoculation. The mean time parameter was fairly well known and constrained in interpretation, because it was based on an empirical inoculation distribution function. The number of deaths solely attributable to COVID-19 inoculation is completely unknown.

As will be shown in the next section, the numbers of deaths reported in VAERS are strongly related to the inoculation date by clustering, but those who died might also have been those who would have died anyway because they were expected to die. There were probably some of each in that group reported. But we have no idea of the total number whose death could be directly attributed to COVID-19 inoculation and who were not in the group expected to die. For all we know, there could have been ten million people in that group, and only an extremely small fraction of that total group was reported in VAERS.

Suppose, for example, that the actual number of deaths reported in VAERS came from two groups: 90 % were from the inoculation-attributable death group and 10 % were from the expected death group. Assume there is no overlap between the two groups. In that case, what VAERS shows is not that 1% of actual expected deaths were reported, but rather that 1/10 of one percent of the expected deaths were reported. If that metric is used as the standard to scale up to total deaths, then the number in the actual inoculation-attributable death group is not 100 times the VAERS reported deaths, but rather 1000 times the VAERS-reported deaths! The point is we can't "reverse-engineer" the reported VAERS death numbers to get the actual inoculation-attributable deaths because it depends on the unknown contribution of each of the two groups (expected deaths and inoculation-attributable deaths) to the VAERS reported deaths, and we can't separate those out.

All this analysis shows is that, at best, only about 1% of the number expected to die was reported, and because the number reported in VAERS included deaths from both groups, the fraction from each actual group of deaths could not be determined. Realistically, we may have to wait until mid-2022, when the 2021 total deaths for each age group are finalized, to ascertain whether we can see increases in all-cause mortality that could have come from the inoculation-attributable deaths.

A1-a3 Local Analysis

Another way of estimating VAERS reporting efficiency is to perform a local analysis, focused on clustering about date of COVID-19 inoculation. For the 65+demographic, the post-inoculation deaths cluster near the vaccination date, providing evidence of a ***strong link to the inoculation.***

Following the approach in the first section of this appendix, we calculate the deaths expected in any ten-day period based on 2019 pre-COVID-19 death statistics. For the inoculated group, the number of deaths expected for any ten-day period are (2,117, 332 deaths/per year) x (44,000,000/54,000,000 fraction of population in age range inoculated) x (10/365 fraction of year), or ~47,270 deaths.

~BEST-CASE SCENARIO

Consider the ten days following inoculation (including day of inoculation). Approximately 2,000 deaths were reported in VAERS. Assume hypothetically that all these deaths were in the expected category; this can be viewed as a *best-case scenario.* In this ~*best-case scenario*, where the concentration of deaths is the highest and is normalized to the expected number of non-COVID-19 inoculation deaths (excluding deaths due solely to COVID-19 inoculation), 2,000/47,270 % of actual deaths (inoculation-related or not), or 4.23%, are reported in VAERS. Thus, at best, VAERS is underreporting by a factor of ~20.

Suppose in that ten-day interval there had been 10,000 deaths that could be directly attributed to COVID-19 inoculation in addition to the expected deaths. This would have given a ratio of 2,000/57,270 actual total deaths, or 3.5 % reported in VAERS. This latter approach requires less assumptions than the former approach, but still yields results of only a few percent actual deaths reported in VAERS.

The Harvard Pilgrim electronic tracking study of post-vaccination events reported to VAERS performed in 2010 [4] showed a 1 % reporting rate for a thirty-day period. In the present case, ~2900 post-inoculation deaths were reported to VAERS within thirty days of inoculation, or ~82 % of total deaths for the 65+demographic. Substituting thirty days for ten in the above computation yields 141,810 expected non-COVID-19 post-inoculation deaths for the thirty-day period, or 2% that are reported in VAERS. The Harvard study used an electronic system that automatically tracked every event that occurred, no matter how small. Because of the effort (time and cost) required to submit event reports to VAERS, we suspect that only the more serious events, such as death, would be reported, and even in this case, the numbers reported are miniscule.

We also did an analysis for sixty days post-inoculation. In the present case, ~3300 post-inoculation deaths were reported to VAERS within sixty days of inoculation, or ~93 % of total deaths for the 65+demographic. Substituting sixty days for ten in the above computation yields 283620 expected non-COVID-19 post-inoculation deaths for the thirty-day period, or 1.2 % that are reported in VAERS. Remember, this normalization is based only on expected deaths. If 100,000 deaths attributable mainly to the COVID-19 inoculation beyond those that overlapped with the expected group occurred during this period, then the denominator would have to be increased by 100,000, yielding a VAERS reporting rate of 0.86 %.

Thus, both the global and local analyses, and the Harvard Pilgrim empirical analysis, are converging on the same two orders-of-magnitude difference between the actual number of deaths that occurred in the USA and those reported in VAERS. Depending on how many people have really died as a result of the COVID-19 inoculation, this reporting rate could well be a fraction of a percent!

A1-a3a Local Clustering Analysis

We end this appendix with one more example from the local analysis. Some background perspective is required. In the buildup to the pandemic (putting aside the issue of high false positives from PCR tests run at high numbers of amplification cycles), almost anyone who died **with** COVID-19 was assumed to have died **from** COVID-19, irrespective of the number of potentially lethal comorbidities they had. The CDC admitted later that about 94 % of the deaths attributed to COVID-19 would ordinarily have been attributed to one of the comorbidities.

For this example, we adopt a similar philosophy for the COVID-19 inoculations. People in the 65+ demographic who have died following inoculation are divided into two groups: those who died **from** the inoculation and those who died as **expected** based on pre-COVID-19 death data. The two groups range from being entirely separate to completely overlapping. We will examine two cases: entirely separate and completely overlapping.

How are the members of each group determined? The death **from** inoculation group consists of those whose deaths cluster significantly around the date of inoculation. The deaths expected group are the number who would have died in the absence of COVID-19. We allow for overlap, where each person who died can be double-valued (a member of both groups), but not double-counted.

To obtain a relatively precise estimate of expected deaths, we would want to select a region of time where the distribution function has substantially leveled off. From Fig. A1, the thirty-sixty-day range appears reasonable. However, there is a time issue here. Given the lag time in data reported by VAERS, most of the data in this range will probably have come from inoculations in January and February, and early-mid March, approximately 35 percent of the total inoculations. Therefore, we could multiply the thirty-sixty-day average number of deaths by ~3 to obtain ~40 expected deaths per day. An even simpler way to estimate the expected deaths reported in VAERS is to use the 15–30-day average shown, which will represent most of the range. This value is ~37, which is close to the ~40 obtained with the above approximation. This analysis should be re-run in three-four months, when more of the long-range data has been filled in.

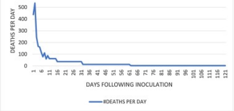

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Fig. A1. Figure A1-1 is a plot of number of deaths from COVID-19 inoculation (reported to VAERS and obtained from the CDC search engine CDC Wonder) as a function of days from inoculation (zero reflects day of inoculation). If there were no effect from the inoculation, as claimed by the CDC and other official government agencies, the curve would be essentially a straight horizontal line, reflecting normal expected deaths in a non-COVID-19 year. The curve is stepped past the tenth day because the data after that point is provided in bands by CDC Wonder. The knee of the curve, which will denote the beginning of the transition of 1) deaths **from** inoculation to 2) deaths **expected**, appears somewhere in the range between day ten and day thirty.

Table A2 shows the results of our analysis. As stated previously, two separate cases were analyzed: completely separate groups and completely overlapping groups. Two values of daily expected deaths were used: the 37 as described above, and 20 to account for potentially lower expected death reporting when the VAERS data has filled in more completely.

Thus, based on the deaths reported in VAERS following COVID-19 inoculation, and assuming the inoculation-related deaths are reported in the same ratio as expected deaths, the actual number of deaths strongly related to the COVID-19 inoculation should be scaled up by factors of 100–200. For the broadest definition of VAERS coverage provided by CDC Wonder, which includes the USA and all territories, protectorates, and possessions, the total deaths following COVID-19 were ~5200 in early June 2021. Using our scaling factors, this translates into somewhere between one-half million and one-million deaths, and this has not taken into account the lag times associated with entering data into VAERS. Compared with the ~28,000 deaths the CDC stated were due to COVID-19 and not associated morbidities for the 65+ age range, the ***inoculation-based deaths are an order-of-magnitude greater than the COVID-19 deaths!*** It should be remembered these are only the **very-short-term inoculation-based deaths**, and could increase dramatically if mid- and long-term adverse effects come to fruition.

We end this appendix with an even more unsettling possibility. The main assumption upon which the results in Table A2 were based is that the post-inoculation temporal distribution function shown in Fig. A1 could be divided into two regions. The strongly varying region originating from the inoculation date reflected deaths from the inoculation, and the essentially flat region that followed reflected expected deaths (that flat region also started at the inoculation date, and formed the base on which the highly varying region is positioned). This model excludes the possibility that deaths from the inoculation extend well beyond the limits of the highly varying region.

Table A2. Actual COVID-19 inoculation-based deaths.

Actual COVID-19 inoculation-based deaths from vaers reporting				
	Separate Groups		Overlapping Groups	
Expected Deaths Reported	37	20	37	20
Range Of Days Inoculation Deaths	0–30	0–30	0–30	0–30
Total Reported Deaths Over Range	2901	2901	2901	2901
Total Expected Deaths Over Range	1147	620	1147	620
Inoculation-Based Deaths Reported	1754	2281	2901	2901
Expected Deaths Reported/Total Expected	.0077	.0041	.0077	.0041
Total Actual Inoculation-Based Deaths Using Expected Ratio (Above)	227792	556341	376753	707561

We know in general this is not true. There can be lag effects such as ADE in the Fall viral season, and longer-term effects such as autoimmune diseases. We postulate that there are other effects from the inoculation that could result in the same flat death profile as that for expected deaths.

Consider the following. Some of the damage we have seen following the inoculations in VAERS includes coagulation/clotting effects and neurological effects of all types [63]. If these effects are not lethal initially, they raise the level of dysfunction. Thus, platelet aggregation has increased to a new base level, and micro-clots have raised the probability of serious clots forming from other lifestyle factors [105]. Death of specific neurons can increase the risk of Alzheimer's disease or Parkinson's disease, and can accelerate the onset of these and many other diseases. Thus, the adverse impacts of the COVID-19 inoculations could be viewed as raising the level of expected deaths in the future. Any deaths of this nature reported in VAERS would need to be viewed as inoculation-driven, and the expected deaths used in the computations would be reduced accordingly.

Consider Table A3 below. The "expected deaths reported" have been reduced below their counterparts in Table A2 to illustrate parametrically how the total inoculation-based deaths would change from VAERS reporting if this baseline effect is operable. While Table A2 used values of 37 and 20 for expected deaths, Table A3 uses values of 10 and 15.

Table A3. Possible COVID-19 inoculation-based deaths.

Possible COVID-19 inoculation-based deaths from vaers reporting				
	Separate Groups		**Overlapping Groups**	
Expected Deaths Reported	10	15	10	15
Range Of Days Inoculation Deaths	0–30	0–30	0–30	0–30
Total Reported Deaths Over Range	2901	2901	2901	2901
Total Expected Deaths Over Range	310	465	310	465
Inoculation-Based Deaths Reported	2591	2436	2901	2901
Expected Deaths Reported/Total Expected	.0021	.0031	.0021	.0031
Total Actual Inoculation-Based Deaths Using Expected Ratio (Above)	1233810	785806	1381429	935806

Thus, if the baseline of the host for coagulation/clotting, inflammation, hypoxia, neurodegeneration, etc., has been raised by the inoculations, translating into an increase in expected deaths and accelerated deaths, then it is entirely plausible that the VAERS death numbers reflect over a million deaths from COVID-19 inoculations so far. These are very short-term-effects only, and time will tell whether the large potential waves of ADE-driven deaths and autoimmune-driven deaths come to pass.

Appendix B

DETAILED ANALYSIS OF MAJOR COVID-19 INOCULANT CLINICAL TRIALS

A2-a Clinical Trials in the Mainly Adult Population

Definitions

Efficacy is the degree to which a vaccine prevents disease, and possibly also transmission, under ideal and controlled circumstances – comparing a vaccinated group with a placebo group [106].

Effectiveness refers to how well a vaccine performs in the real world [107]

Relative Risk (RR) is computed by dividing the percentage of patients that contracted disease in the vaccine arm by the percentage of patients that contracted disease in the placebo arm.

Relative Risk Reduction (RRR) is computed by subtracting the RR from 1.

Absolute Risk Reduction (ARR) is computed by subtracting the percentage that contracted disease in the vaccine arm from the percentage that contracted disease in the placebo arm.

Absolute Risk = probability = incidence.

Cumulative Incidence represents the number of new cases in a period of time / population at risk.

Incidence Density is the number of new cases of a given disease during a given period in specified population; also, the rate at which new events occur in a defined population.

Immunogenicity is the ability of a molecule or substance to provoke an immune response or the strength or magnitude of an immune response. It can be a positive (wanted) or negative (unwanted) effect, depending on the context.

Immune Response is an integrated systemic response to an antigen (Ag), especially one mediated by lymphocytes and involving recognition of Ags by specific antibodies (Abs) or previously sensitized lymphocytes [108]

Safety data for Pfizer and Moderna trials:

There were two major COVID-19 inoculant clinical trials: Pfizer/BioNTech and Moderna.

The Pfizer clinical trials were titled officially "a phase 1/2/3, placebo-controlled, randomized, observer-blind, dose-finding study to evaluate the safety, tolerability, immunogenicity, and efficacy of sars-cov-2 rna vaccine candidates against covid-19 in healthy individuals" [98]. The "Actual Study Start Date" was 29 April 2020, the "Estimated Primary Completion Date" was 2 November 2020, and the "Estimated Study Completion Date" is 2 May 2023. Thus, the mass inoculation rollout so far has been conducted in parallel with the Pfizer Phase III Clinical Trial. For all practical purposes, the mass global inoculation of the Pfizer inoculant recipients can be considered Phase III 2.0 of the Clinical Trials! The inclusion criteria for the official Phase III Clinical Trials incorporated (as stated in the title and in the protocol document) healthy individuals, while the criteria for mass inoculation went well beyond healthy individuals. In essence, we have an official Phase III Clinical Trial with ~43,000+ healthy individuals, and an unofficial Phase III Clinical Trial with billions of individuals covering a wide spectrum of health levels [98].

The Pfizer Phase III trials were initiated July 2020, the efficacy data were submitted to the FDA for EUA approval in November 2020, and FDA approval was granted in December 2020. Six deaths occurred in the Pfizer trial, two in the inoculated group and four in the placebo group (which received saline) [33]. The two inoculated, both over the age of 55, died of cardiovascular causes. One died three days after inoculation and the other died 62 days after inoculation [109]. These two deaths were comparable (in frequency and cause) to placebo group deaths and perhaps more importantly, similar to the general population at that age. In the case of Moderna, there were 13 deaths, six in the inoculated group, seven in the placebo group (normal saline placebo, a mixture of sodium chloride in water 0.90 % w/v) at 21–57 days after the inoculation ([103]b).

In a report by the Norwegian National Medicines Association, published on 15 January 2021, there were 23 elderly people (all over the age of 75 and frail) in

January 2021, there were 23 elderly people (all over the age of 75 and frail) in nursing homes, who died at various intervals from the time of inoculation with mRNA inoculant The report then suggested that, following the assessment, 13 of the 23 deaths would have been a direct result of the side effects of inoculation. It is possible that the other 10 deaths were post-inoculation, but not directly related to side effects, so not necessarily related to the inoculant itself [109].

It is no surprise that frail elderly people can be fatally destabilized by adverse reactions associated with post-inoculation inflammation, which in a young adult would have been considered minor. It is also no surprise that frail elderly people with comorbidities can be fatally destabilized from COVID-19 infection, which in a young adult or child would have been considered minor. A frail elderly person can be fatally destabilized by a simple coughing fit! This does not mean that these deaths are not events that need to be taken very seriously; on the contrary, if confirmed, they should guide inoculation policies in this category of patients from now on. Specifically, each case should be carefully assessed and an inoculation decision made based on the risk-benefit ratio [110].

In light of these data, the question may arise as to why there were no inoculant-attributed deaths in clinical testing of inoculants. The answer is that neither Pfizer nor Moderna included frail patients and included only a small number of very elderly patients - those over 75 accounted for 4.4 % of the total tested for Pfizer and 4.1 % for Moderna. While they could not in fact determine a causal relationship between inoculation and death, they also could not rule out that the inoculations had accelerated the deterioration of the condition of those patients [33].

Effectiveness data

As defined previously, the effectiveness of a vaccine lies in its ability to prevent a particular disease. If designed, tested, and administered correctly, authorized vaccines are effective in preventing disease and protecting the population. Like medicines, vaccines are not 100 % effective in all vaccinated people. Their effectiveness in a person depends on several factors. These include: age; other possible diseases or conditions; time elapsed since vaccination; previous contact with the disease.

To be declared safe and effective, a vaccine against COVID-19 infection must pass a series of tests and must meet regulatory standards, like any other vaccine or drug approved on the pharmaceutical market [111].

Regarding Pfizer and Moderna trials:

The first important note is that maximum efficiency does not come immediately, because the immune response needs time.

In the case of Pfizer, the chance of developing COVID-19 becoming virtually the same between the inoculated and placebo groups increases up to 12 days after the first inoculation, then gradually decreases for those inoculated. The inoculum efficiency between the first and second doses is 52 % [106], but it is unclear what long-term protection a single dose provides. After the second dose, the effectiveness rises to 91 % and only beyond 7 days after the second dose is 95 % reached. However, the ARR for the latter case is only 0.7 % [112]. In other words, within 12 days after the first dose we can get COVID-19 as if we had not been inoculated. Another important aspect is that we still do not know if the Pfizer inoculant prevents severe cases. Seven days after the second dose, there were four severe cases of COVID-19, one in the inoculated group and three in the placebo group, which is far too low for us to make a statistical assessment. There are as yet no data on the inoculant's ability to prevent community transmission. Realistically, the effectiveness of the inoculant in preventing asymptomatic cases has not been tested.

For Moderna, the effectiveness is only 50 % in the first 14 days after the first dose and reaches a maximum of 92.1 % on the edge of the second dose (ARR of 1.1 %, which is 28 days, not 21 as in the case of Pfizer) [46]. Moderna also did not test the long-term efficacy of a single dose. Then, 14 days after the second dose, the effectiveness rises to 94.1 %, with the amendment being an average. Thus, in people over 65 it was 86.4 %, compared to 95.6 % in the 18–65 age range ([103]). It is a minor difference from Pfizer, which declares equal efficiency in all age groups. An important observation is the statement by Moderna that their inoculant prevents severe cases, but only more than 14 days after both doses [126]. All 30 severe cases were in the placebo group, suggesting 100 % efficacy. After a single dose, there were two severe cases among those inoculated and four in the placebo group [33]. Last, but not least, unlike Pfizer, Moderna tested the presence of asymptomatic infection by RT-PCR before the second dose: there were 39 asymptomatic cases in the placebo group and 15 in the inoculated group. It is difficult to draw definitive conclusions due to the small number of cases. These data suggest that the inoculant reduces, but does not prevent, asymptomatic transmission [126].

A2-b Ongoing Clinical Trials in the Pediatric Population

In a recent Phase III study performed in the pediatric population, Comirnaty (Pfizer) was tested on a group of 2,260 children, aged 12–15, years who had no previous clinical signs of SARS-CoV-2 infection. They were divided into two groups, one placebo (978 children) and the other with Comirnaty (1005 children). In the Comirnaty group, of the 1005 children in whom the serum was administered, none developed COVID-19 disease, compared with the placebo group in which 16 children in 978 had clinical signs of the disease. The Pfizer study showed that the children's immune response was comparable to the immune response in the 16–25 age group (measured by the level of antibodies against SARS-CoV-2). It could be concluded that in this study, Comirnaty was 100 % effective in preventing SARS-CoV-2 infection, although the actual rate could be between 75 % and 100 %. [63]. The results will be evaluated by the FDA and EMA.

The predictive value (for mass inoculation results) of the Comirnaty trial for the children aged 12–15 years is questionable. There were 1005 children who were inoculated with Comirnaty. Using the rule of three in statistics, where to obtain a predictive result of 1/x with high confidence (e.g., 1 in a thousand), 3x participants are required for the test sample. For the Comirnaty test sample of 1005, an adverse event of about 1/340 could be detected with high confidence.

What does this mean in the real world? In the USA, there are approximately 4,000,000 children in each age year for adolescents. Thus, there are ~16,000,000 children in the 12–15 age band. A serious adverse event, including death, that occurred at a 1/800 rate would not be detectable with high confidence in a sample of 1005 people. Thus, the results of the trials for 1005 children would allow for 20,000 children to suffer a non-trial-detected serious adverse event, including death, when extrapolated to potential inoculation of all children in the 12–15 age group! Given that the risk of contracting COVID-19 with serious outcomes is negligible in this population, ***proceeding with mass inoculation of children 12–15 years old based on the trials that were conducted cannot be justified on any cost-benefit ratio findings.***

Also, the evaluation of efficacy in children aged 6 months to 11 years has recently begun and continues [24]. Pfizer began enrolling children under 12 to evaluate the COVID-19 mRNA inoculant. Also, Comirnaty will be evaluated in a new clinical trial for children aged 6 months to 11 years. In the first phase, the study will enroll 144 people and will identify the required dose for 3 age groups (6 months - 2 years, 2–5 years and 5–11 years). After a 6-month follow-up period, the parents/guardians of children in the placebo group will have the option of allowing their children to receive the inoculation. The results are expected in the second half of 2021.

Moderna also began a study to evaluate the mRNA inoculation in children aged 6 months to 12 years. Both companies have already started testing vaccines in 14-year-olds. In the US, children make up 23 % of the population [113].

Data on the risks and benefits of possible inoculation in children and adolescents are currently insufficient and no recommendation can be made. Specifically, mass child inoculations cannot be recommended until the benefits and minimal projected risks have been demonstrated in a sufficiently large trial to provide confidence that mass inoculation will have an acceptable level of adverse effects relative to the demonstrated benefits. On the other hand, children often experience COVID-19 asymptomatically, and the SARS-CoV-2 infection progresses harmlessly. Currently, in the context of limited inoculation capacities, there is no indication of urgent inoculation of children. In the context of declining incidences of SARS-CoV-2 infections and demonstrated low serious adverse effects from COVID-19 infections for children and adolescents, the issue of inoculating children and adolescents is no longer paramount. Authorized forums must calculate what prevails for children and adolescents: the benefits or risks.

A2-c Clinical Trial Issues for Other Categories

Although people with severe comorbidities such as obesity or oncological conditions were not initially included in the clinical trials that led to obtaining EUA, they were included in subsequent studies, some even ongoing. In their case, it seems that the efficacy was lower compared to the results obtained initially with healthy adults.

The interim analysis of data from a prospective observational study indicates the need to prioritize cancer patients for timely (respectively 21-day) booster administration in the case of administration against COVID-19 with Comirnaty. According to the study, the effectiveness of a single dose of Comirnaty among cancer patients is low, but the immunogenicity of patients with solid cancers increased at 2 weeks after receiving the second dose of inoculant 21 days after the first dose. Because the study was conducted in the UK, participants inoculated before December 29, 2020 received two doses of Comirnaty 21 days apart, and those who started the regimen after this date were scheduled to receive a second dose of Comirnaty 12 weeks apart. first administration. Thus, the study continues to collect data from participants receiving Comirnaty 12 weeks after the first dose.

Approximately 21 days after a single dose of Comirnaty, the proportion of study participants who tested positive for anti-S IgG antibodies was [114]:

94 % among healthy participants;

38 % among patients with solid cancers;

18 % among patients with hematological cancers.

Among participants who received the 21-day booster and for whom biological samples were available two weeks after the second dose, the following proportions of confirmation as seropositive for anti-S IgG antibodies were reported [114].

100 % of healthy participants, compared to 86 % of the same group of participants who did not receive the second dose;

95 % of patients with solid cancers, compared with 30 % of the same group of participants who did not receive the second dose;

60 % of patients with hematological cancers, compared with 11 % of the same group of participants who did not receive the second dose.

Two other studies suggest low immunogenicity in the context of Comirnaty administration in patients with hematological cancers. In one study, patients with chronic lymphocytic leukemia (CLL) had significantly reduced immune response rates to COVID-19 inoculation compared to healthy participants of the same age. Considerable variations in post-administration immune response have been reported among patients with CLL depending on their stage of treatment

The effectiveness of Comirnaty administration was also evaluated in elderly patients with multiple myeloma [115]. 21 days after administration of the first dose of Comirnaty inoculation (before receiving the second dose), 20.5 % of patients with multiple myeloma compared to 32.5 % of control participants had neutralizing antibodies against SARS-CoV-2. One possible explanation could be that the therapy negatively affects the production of antibodies. However, the administration of the second dose is important for the development of the immune response in these patients [115].

Preliminary data from the v-safe surveillance system, the v-safe pregnancy registry and the Vaccine Adverse Event Reporting System (VAERS) do not indicate obvious safety signals regarding pregnancy or the associated neonatal implications with mRNA injections against COVID-19 *in the third trimester of pregnancy* [3]. The study included 35,691 pregnant women [116]. Compared to non-pregnant women, pregnant women reported more frequent pain at the injection site as an adverse event associated with mRNA COVID-19 vaccination, and headache, myalgia, chills, and fever were reported less frequently. In the context where initial clinical trials of messenger RNA-based inoculants have not evaluated the efficacy and safety of innovative technology among pregnant women, these preliminary data *from the third trimester only* help to inform both pregnant women and health professionals in making the inoculation decision. However, continuous monitoring through large-scale longitudinal studies remains necessary to investigate the effects associated with maternal anti-COVID-19 inoculation on mothers, pregnancies, the neonatal period and childhood.

On the other hand, the inoculation landscape has become even more complex due to new circulating viral variants. Authorities recommend genomic surveillance and adaptation in order to be effective against new variants (different from the initial strain that was detected at the end of 2019). The efficacy data of Comirnaty against circulating viral variants are highlighted in a very recent study in Israel which showed that the protection offered by the Pfizer inoculant against variant B.1.351 (first identified in South Africa) is lower [112].

The results have not yet been submitted to the expertise of specialists. The study compared nearly 400 adults who were diagnosed with COVID-19 at least 14 days after receiving one or two doses of the inoculant to the same number of uninoculated people. It was found that B.1.351 represents approximately 1 % of the COVID-19 cases studied. But among patients who received two doses of inoculant, the prevalence rate of the variant was eight times higher than in those not inoculated - 5.4 % compared to 0.7 %. This suggests that Comirnaty is less effective against variant B.1.351, compared to the original variant and variant B.1.1.7. The limitation of the study comes from the small number of adult people studied, but it is an alarm signal for a closer study of these cases. In addition, it seems that at present, the prevalence of this variant is low. On the other hand, in early April, Pfizer announced that according to the results of the Phase III study in the adult population, Comirnaty also demonstrated 100 % efficacy in the prevention of Covid-19 disease caused by SARS-CoV-2 variant B.1.351 (9 cases of Covid-19 were recorded, all in the placebo group, and after sequencing it was found that 6 had been determined by B.1.351) [117].

Appendix C

MID- AND LONG-TERM ADVERSE EFFECTS FROM PRIOR VACCINES

A 2020 study emphasizing mid- and long-term adverse effects from prior vaccines [4] identified the following sixteen mid- and longer-term potential issues concerning vaccines. These include:

3.1. Antibody-Dependent Enhancement (where enhanced virus entry and replication in a number of cell types is enabled by antibodies);

-1a. *Intrinsic Antibody-Dependent Enhancement* (where non-neutralizing antibodies raised by natural infection with one virus may enhance infection with a different virus);

-1b. *Immune Enhancement* (enhancement of secondary infections via immune interactions);

-1c. *Cross-Reactivity* (an antibody raised against one specific antigen has a competing high affinity toward a different antigen.);

-1d. *Cross-Infection Enhancement* (infection enhancement of one virus by antibodies from another virus);

3. 2. Vaccine-Associated Virus Interference (where vaccinated individuals may be at increased risk for other respiratory viruses because they do not receive the non-specific immunity associated with natural infection);

3. *Vaccine-Associated Imprinting Reduction* (where vaccinations could also reduce the benefits of 'imprinting', a protection conferred upon children who experienced infection at an early age)

4. *Non-Specific Vaccine Effects on Immune System* (where previous infections can alter an individual's susceptibility to unrelated diseases);

5. *Impact of Infection Route on Immune System* (where immune protection can be influenced by the route of exposure/delivery);

6. *Impact of Combinations of Toxic Stimuli* (where people are exposed over their lifetime to myriad toxic stimuli that may impact the influence of any vaccine);

7. *Antigenic Distance Hypothesis* (negative interference from prior season's influenza vaccine (v1) on the current season's vaccine (v2) protection may occur when the antigenic distance is small between v1 and v2 (v1 ≈ v2) but large between v1 and the current epidemic (e) strain (v1 ≠ e).);

8. *Bystander Activation* (activation of T cells specific for an antigen X during an immune response against antigen Y);

9. *Gut Microbiota* (Impact of gut microbial composition on vaccine response);

10. *Homologous Challenge Infection Enhancement* (the strain of challenge virus used in the testing assay is very closely related to the seed virus strain used to produce the vaccine that a subject received);

11. *Immune Evasion* (evasion of host response to viral infection);

12. *Immune Interference* (interference from circulating antibody to the vaccine virus);

12a. *Original Antigenic Sin* (propensity of the body's immune system to preferentially utilize immunological memory based on a previous infection when a second slightly different version of that foreign entity (e.g. a virus or bacterium) is encountered.);

13. *Prior Influenza Infection/Vaccination* (effects of prior influenza infection/vaccination on severity of future disease symptoms);

14. *Timing between Viral Exposures* (elapsed time between viral exposures);

15. *Vaccine-Associated Enhanced Respiratory Disease* (where vaccination enhances respiratory disease); and

16. *Chronic Immune Activation* (continuous innate immune responses).

Most of these events are not predictable, and most, if not all, would be possible for the COVID-19 inoculant in the mid- and long-term for adults and children.

3.3. Mid- and Long-Term Serious Illnesses for Adults and Children from Past Vaccines

As stated in the aforementioned 2020 study on vaccine safety: "The biomedical literature is very sparse with studies on long-term vaccine effects, especially long-term adverse effects. Large numbers of people and long periods of time are required to identify such adverse events, and draw statistically-valid connections between vaccinations and disease. These efforts would be very resource-intensive, and there appears to be little motivation among the vaccine producers and regulators to make these resources available for such studies. Thus, the following examples reflect the extremely small tip of an extremely large iceberg of long-term adverse vaccine effects." [4]

"The two main categories of diseases reported in the biomedical literature triggered by past vaccinations are "Autoimmune (e.g., Systemic Lupus Erythematosus, Psoriasis, Arthritis, Multiple Sclerosis, Hepatitis, Uveitis, Pseudolymphoma, Guillain-Barre Syndrome, Thrombocytopenic Purpura, etc.) and Neurological (e.g., Central Demyelinating Diseases, Developmental Disability, Febrile seizures, Narcolepsy, Encephalomyelitis, Autonomic Dysfunction, etc.). Others include Diabetes, Gastrointestinal, Joint-related, Necrobiotic Granuloma, Neutropenia, Pulmonary Fibrosis, etc."

"Vaccinations may also contribute to the mosaic of autoimmunity [118]. Infrequently reported post-vaccination autoimmune diseases include systemic lupus erythematosus, rheumatoid arthritis, inflammatory myopathies, multiple sclerosis, Guillain-Barre syndrome, and vasculitis".

"Studies have demonstrated a latency period of years between HiB vaccination and diabetes mellitus, and between HBV vaccination and demyelinating events [118] latency periods can range from days to years for postinfection and postvaccination autoimmunity".

"Most of the extra cases of IDDM appeared in statistically significant clusters that occurred in periods starting approximately 38 months after immunization and lasting approximately 6–8 months. Immunization with pediatric vaccines increased the risk of insulin diabetes in NOD mice.Exposure to HiB immunization is associated with an increased risk of IDDM." [4]

Thus, even the sparse past vaccine studies that went beyond the short-term showed latency effects of serious diseases occurring ***three years or more*** post-vaccination.

Appendix D

COST-BENEFIT ANALYSIS OF COVID-19 INOCULATIONS

This appendix presents a non-traditional *best-case scenario* pseudo-cost-benefit analysis of the COVID-19 inoculations for the 65+ demographic in the USA. In this incarnation of a cost-benefit analysis, the costs are the number of deaths resulting from the inoculations, and the benefits are the lives saved by the inoculations. The time range used was from December 2019 to end-of-May 2021.

It is assumed, in this best-case scenario, that all the deaths truly attributable to COVID-19 only could have been eliminated by the inoculations given (about half the USA population has been inoculated at this time) [88,119]. It can be conceptualized as the vaccines having been available in Summer 2019, and subsequent administration having eliminated all the deaths experienced that were truly attributable to COVID-19. If the cost-benefit ratio is ***poor*** for this *best-case scenario*, it will be ***very poor*** for any real-world scenario [120].

We will use Fig. 1, Fig. 2 as starting points to conduct a cost-benefit analysis of

COVID-19 inoculations for the most vulnerable demographic, those 65 + . We start with the official government numbers for COVID-19 and post-inoculation deaths, and modify them to arrive at actual deaths resulting from COVID-19 and the inoculations. We compare the two numbers (appropriately normalized) to ascertain costs vs benefits .

As Fig. 1 shows, there are three age bands that comprise the 65+ demographic. We weight the COVID-19 deaths per capita in each band by the band's population, and divide the sum of these three products by the total 65+ population to arrive at an average COVID-19 deaths per capita of 0.0087 for the total 65+ demographic.

Fig. 2 contains two normalizations. First, the deaths were normalized by total inoculations given, not by people inoculated or people who had completed the full series of inoculations. We will retain the normalization by total inoculations given, since it will provide the ***most conservative results*** (largest denominator) for estimation purposes. Second, the deaths were normalized/restricted to those occurring within seven days post-inoculation. This normalization was done to compare across age bands, where the inoculations started at very different points in time. For the present cost-benefit purpose, where we are concentrating on the 65+ band, we remove this latter normalization, and include all post-inoculation deaths. Removing this normalization increases deaths per inoculation by about 40 % to a value of 0.000032, and offers a more credible comparison to the numbers from Fig. 1.

Thus, based on the CDC's official numbers, there are an average COVID-19 deaths per capita of 0.0087 and an average deaths per inoculation of 0.000032 for the 65+ demographic. The chances of a person 65+ dying from an inoculation relative to their chances of dying from COVID-19 are approximately 0.0037, or about 1/270, based on these official CDC figures.

However, as we have shown previously, three corrections to these numbers are required to convert them to real-world effects. First, as the Harvard Pilgrim study has shown and as our results in Appendix 1 confirm, VAERS is underreporting actual deaths by about two orders of magnitude. Applying this correction alone to the above 1/270 ratio changes the risk benefit to about 1/3., Second, as the CDC has stated, approximately 94 % of the COVID-19 deaths could have been attributed to any of the comorbidities these patients had, and only 6% of the deaths could actually be attributed to COVID-19. As we pointed out, if pre-clinical comorbidities had been included, this number of 6% would probably be decreased further. For ***conservative*** purposes, we will remain with the 6%. Applying this correction to the 1/3 risk-benefit ratio changes it to 5/1! Third, as a comprehensive survey of false positives from RT-PCR tests concluded: "evidence from external quality assessments and real-world data indicate enough a high enough false positive rate to make positive results highly unreliable over a broad range of scenarios" [127]. Because of the myriad RT-PCR tests performed in the USA to screen for/diagnose COVID-19 using different values for Ct and different procedures, a specific number for false positives cannot be obtained at this point in time. Again, these false positives would reduce the 6% number, perhaps substantially. And again, for ***conservative*** purposes, we will remain with the 6% number.

Thus, our ***extremely conservative*** estimate for risk-benefit ratio is about 5/1. In plain English, people in the 65+ demographic are five times as likely to die from the inoculation as from COVID-19 under the most favorable assumptions! This demographic is the most vulnerable to adverse effects from COVID-19. As the age demographics go below about 35 years old, the chances of death from COVID-19 become very small, and when they go below 18, become negligible.

It should be remembered that the deaths from the inoculations shown in VAERS are short-term only (~six months for those inoculated initially), and for children, extremely short-term (~one month) [3]. Intermediate and long-term deaths remain to be identified, and are possible from ADE, autoimmune effects, further clotting and vascular diseases, etc., that take time to develop. Thus, the long-term cost-benefit ratio under the *best-case scenario* could well be on the order of 10/1, 20/1, or more for all the demographics, increasing with decreasing age, and an order-of-magnitude higher under real-world scenarios! In summary, the value of these COVID-19 inoculations is not obvious from a cost-benefit perspective for the most vulnerable age demographic, and is not obvious from any perspective for the least vulnerable age demographic.

Appendix Da

PROBLEMS WITH TEST CRITERIA FOR DETERMINING COVID-19

Consider the criteria for determining whether an RT-PCR test result is positive for SARS-CoV-2. The CDC instruction (until 1 May 2021) specifies running the RT-PCR tests for 45 amplification cycles. Then, to interpret the data: when all controls exhibit the expected performance, a specimen is considered positive for SARS-CoV-2 if all SARS-CoV-2 marker (N1, N2) cycle threshold growth curves cross the threshold line within 40.00 cycles (< 40.00 Ct). The RNase P may or may not be positive as described above, but the SARS-CoV-2 result is still valid ([103]a).

Many false positives are possible in the upper part of this cycle threshold range, especially in areas of low prevalence. In particular, virus culture has been found to be unfeasible in cases with a Ct value exceeding 33. A prospective cohort study involving the first 100 COVID-19 patients in Singapore also showed that attempts to culture the virus failed in all PCR-positive samples with a Ct value >30" [121]. During mass testing in Germany, it was found "that more than half of individuals with positive PCR test results are unlikely to have been infectious" [122]. Another study found that tests with low specificity (deriving from use of many cycles) cannot provide strong evidence for the presence of an infection [123]. A systematic review of PCR testing concluded "Complete live viruses are necessary for transmission, not the fragments identified by PCR. Prospective routine testing of reference and culture specimens and their relationship to symptoms, signs and patient co-factors should be used to define the reliability of PCR for assessing infectious potential. Those with high cycle threshold are unlikely to have infectious potential." [89].

As skeptics have argued, in the buildup of the pandemic, the rapid increase in numbers of COVID-19 cases was due in part to the high values of cycle threshold used in the tests. Unfortunately, the true numbers of false positives will probably be unobtainable if an audit were performed, since these values are not reported with the test results: all currently-available nucleic acid tests for SARS-CoV-2 are FDA-authorized as qualitative tests, and Ct values from qualitative tests should never be used to direct or inform patient management decisions. Therefore, it is not good for laboratories to include Ct values on patient reports [124].

After mass inoculations started, a large number of "breakthrough" cases emerged, and a total of 10,262 SARS-CoV-2 vaccine breakthrough infections had been reported from 46 U.S. states and territories as of April 30, 2021 [18]; the number of reported COVID-19 vaccine breakthrough cases is likely a substantial undercount of all SARS-CoV-2 infections among fully vaccinated persons. The national surveillance system relies on passive and voluntary reporting, and data might not be complete or representative. Many persons with vaccine breakthrough infections, especially those who are asymptomatic or who experience mild illness, might not seek testing [18].

This negative outcome of increased "breakthrough" cases motivated the CDC to change a number of reporting and test procedures and issue new regulations for identifying and investigating hospitalized or fatal vaccine breakthrough cases starting 1 May 2021, stating: "For cases with a known RT-PCR cycle threshold (Ct) value, submit only specimens with Ct value ≤28 to CDC for sequencing. (Sequencing is not feasible with higher Ct values.)". Thus, the Ct values for sequencing were lowered from the high false positive range allowed during the pandemic buildup to a limit that would eliminate many of these false positives in the 'breakthrough case' identification phase [101].

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