

Mass Vaccination Against SARS-CoV-2 Is Unjustified

CoV-anon

2021-12-17

Introduction

In this paper, I outline the following argument and provide evidence for each of its premises:

Premise 1: COVID-19 is not particularly deadly for most people given that (1) the risk of COVID-19 mortality is extremely age/morbidity-specific, and (2) deaths attributed to COVID-19 have been substantially overreported (at least in western countries).

Premise 2: Naturally acquired immunity against infection by SARS-CoV-2 is widespread and highly protective against symptomatic reinfection.

Premise 3: The currently available SARS-CoV-2 vaccines are insufficiently tested, relatively poor quality, and unsafe for at least some demographics.

Premise 4: If COVID-19 is not particularly deadly for most people, if naturally acquired immunity against infection by SARS-CoV-2 is widespread and highly protective, and if the currently available SARS-CoV-2 vaccines are insufficiently tested, relatively poor quality, and unsafe for at least some demographics, then mass vaccination (let alone mandatory vaccination) is unjustified.

Conclusion: Mass vaccination (let alone mandatory vaccination) is unjustified.

Premise 1: COVID-19 is not particularly deadly for most people

1. Official Data and Overreporting

COVID-19 mortality is highly age/morbidity-specific. UK data indicate that individuals aged 18 years or less die from or with COVID-19 “at a rate of about 2 for every million people in this age range” (Ledford, 2021). Hence, for individuals aged 18 and under, the absolute risk of dying from or with COVID-19, according to these data, is 1 in 500,000, or, 0.0002%. Similarly, in their study of SARS-CoV-2 infection among children in Germany, Sorg et al. (2021) found that for children with no comorbidities, “the ICU admission rate was 0.2 per 10,000.” The researchers also note that the case fatality ratio for this age group “could not be calculated, due to an absence of cases [i.e., there were too few deaths in this age range to make a precise calculation]” (Sorg et al., 2021). Broader analyses draw the same conclusions. In an analysis of 23 seroprevalence surveys (i.e., surveys estimating the number of infections in a given population based on serological tests) representing 14 different countries, Axfors and Ioannidis (2021) found that the median infection-fatality ratio (IFR) (i.e., the number of confirmed deaths divided by the number of infections, averaged out over specific demographics) was 0.0027% for individuals aged 0–19. Even for those aged 60–69, Axfors and Ioannidis (2021) found that the median IFR was 0.59%.

These figures indicate a low level of risk for the general population and for younger cohorts in particular. Indeed, according to data from the United States, 95% of all decedents had an *average of 4 additional comorbidities* or causes of death aside from COVID-19 listed on their death reports (Kostoff et al., 2021)! Hence, it is no wonder that SARS-CoV-2 has been able to spread so efficiently across the globe without wrecking everyone’s health in its wake. All seroprevalence data from Europe and the Americas indicate that “SARS-CoV-2 infection has been very widely spread globally” (Ioannidis, 2021). Indeed, based on seroprevalence data, researchers concluded that somewhere in the order of 2 *billion* infections occurred as of February, 2021, with a global average IFR of 0.15%, which is in-line with the global average IFR of seasonal influenza viruses (Ioannidis, 2021).

When taking these figures into account, we must also consider that they capture the risk of dying from *or with* COVID-19, the latter meaning (in many cases) dying due to some other cause, but only after *incidentally* testing positive for mild or asymptomatic SARS-CoV-2 infection. Consider the following:

“Many UK health spokespersons have been careful to repeatedly say that the numbers quoted in the UK indicate death *with the virus*, not death *due to the virus* – this matters. When giving evidence in parliament a few days ago, Prof. Neil Ferguson of Imperial College London said that he now expects fewer than 20,000 Covid-19 deaths in the UK but, importantly, two-thirds of these people would have died anyway. In other words, he suggests that *the crude figure for ‘Covid deaths’ is three times higher than the number who have actually been killed by Covid-19*. (Even the two-thirds figure is an estimate – it would not surprise me if the real proportion is higher.) [my emphasis].” (Lee, 2020).

Likewise, in the United States, “the federal government requires hospitals to report every patient who tests positive for COVID, yet the overall tallies of COVID hospitalizations, made available on various state and federal dashboards and widely reported on by the media, do not differentiate based on severity of illness.” (Zweig, 2021). Equating deaths and hospitalizations *from* COVID-19 with those that are unrelated to symptomatic illness, but nevertheless occurred after an individual tested positive for SARS-CoV-2 (albeit incidentally), inflates the risks of SARS-CoV-2 infection and biases our perceptions. To take just one example of how conflating symptomatic illness with positive test results biases our perceptions, consider recent overblown reports about children being hospitalized with COVID-19. CNN (2021) and other media outlets frequently run provocative headlines, such as “Every day, hundreds of kids are getting hospitalized with Covid-19. That’s not the only reason to protect kids from Delta, doctors say.” Nevertheless, despite these provocative headlines, reported hospitalization rates “greatly overestimate the true burden of COVID-19 disease in children” (Zweig, 2021). Kushner et al. (2021), in their study of pediatric COVID-19 hospitalizations in San Francisco, found the following:

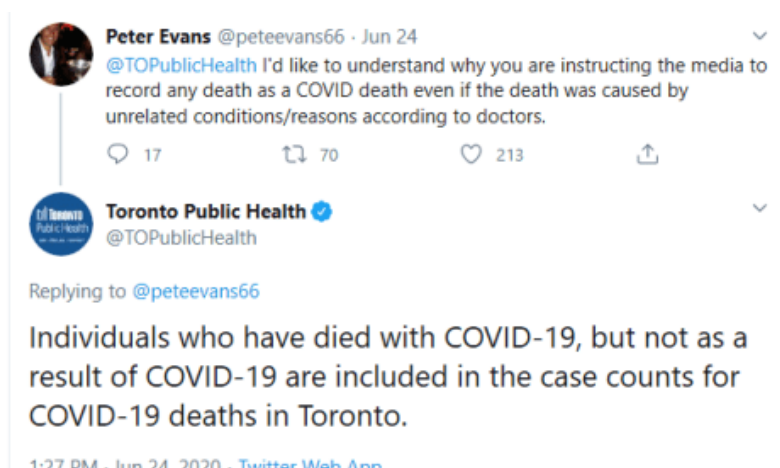
“Of 117 hospitalizations, 46 (39.3%) were asymptomatic, 33 (28.2%) had mild to moderate disease, 9 (7.7%) had severe illness, and 15 (12.8%) had critical illness (weighted κ : 0.82). A total of 14 (12%) patients had multisystem inflammatory syndrome in children. *A total of 53 (45%) admissions were categorized as unlikely to be caused by SARS-CoV-2* (κ : 0.78) [my emphasis].” (Kushner et al., 2021).

If we extrapolate from these figures, we will need to conclude that half of all pediatric hospitalizations coded as ‘COVID-19 hospitalizations’ (and, by extension, those coded as ‘COVID-19 deaths’) are misclassified. These patients only incidentally tested positive for SARS-CoV-2 infection before or while hospitalized for some other reason. Indeed, this assessment agrees with National Health Services (NHS) data from England showing that 56% of all COVID-19 patients in hospital were diagnosed *post-admission* (Donnelly and Yorke, 2021). These patients were admitted to hospital for reasons unrelated to COVID-19. Then, they incidentally tested positive for SARS-CoV-2 infection as a result of being subject to routine testing protocols. Then, they were added to the official COVID-19 hospitalization data that are reported to the media, despite presenting with few or no symptoms and despite being initially admitted for reasons unrelated to COVID-19 (Donnelly and Yorke, 2021).

When attempting to understand the sheer scope of the overreporting of COVID-19 hospitalizations and mortality, we should consider that the US Centre for Disease Control and Prevention (CDC), since March, 2020, has recommended the following protocols to physicians when coding deaths ‘due to COVID-19’:

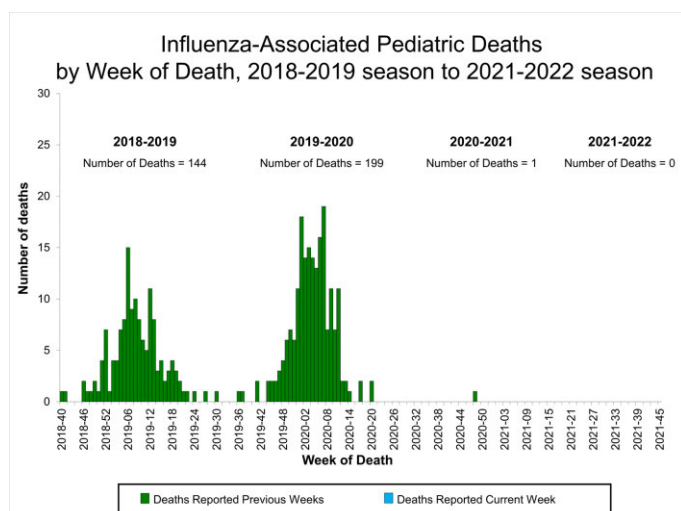
“It is important to emphasise that Coronavirus Disease 19, or Covid-19, *should be reported for all decedents where the disease caused or is presumed to have caused or contributed to death* ... In cases where a definite diagnosis of COVID-19 cannot be made [i.e., no testing whatsoever can be performed], but it is suspected or likely (e.g., the circumstances are compelling within a reasonable degree of certainty), it is acceptable to report COVID-19 on a death certificate as “probable” or “presumed.” In these instances, certifiers should use their best clinical judgement in determining if a COVID-19 infection was likely [my emphasis].” (CDC, 2020).

These protocols, coupled with the mass screening of asymptomatic individuals, have clearly led to substantially overestimated death and hospitalization figures. Many other countries adhere to similar policies. Northern Ireland’s HSC (2021) Public Health Agency releases weekly mortality reports that define a death *due to COVID-19* (i.e., symptomatic disease) as follows: “Individuals who have died within 28 days of first positive [RT-PCR test] result, *whether or not COVID-19 was the cause of death* [my emphasis].” In other words, any person who dies within 28 days of testing positive for SARS-CoV-2, no matter the actual cause of death, will be counted amongst those who died *because of* COVID-19, thereby biasing the data and inflating the mortality/hospitalization risks that are relayed by the media to the general population. The result of this type of reporting is unnecessary fear amongst the populace. Similarly, in the following quote, the advisor to Italy’s health minister describes how the Italian government handles COVID-19 death certificates: “The way in which we code deaths in our country is very generous in the sense that *all the people who die in hospitals with the coronavirus are deemed to be dying of the coronavirus* [my emphasis].” (Newey, 2020). In Canada, our policies are no different. Consider the following:

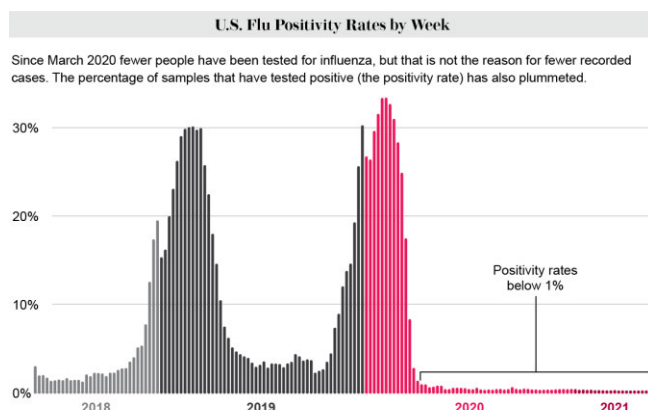


(Toronto Public Health, 2020)

For an estimate of the frequency at which COVID-19 mortality is overreported in western countries, view the following timestamped video: (https://youtu.be/B_ehqHQOBO0?t=1106) (Prof. Manuel Schabus, 2021). In most western European countries and North American countries, COVID-19 deaths have been either tremendously or substantially overcounted (Prof. Manuel Schabus, 2021). We can see the hallmarks of this overreporting and misclassification in other datasets as well. For instance, it is easy to see why presuming COVID-19 as the cause of death when patients merely present with symptoms of common viral respiratory diseases (VRDs) would underestimate deaths due to seasonal influenza viruses and thereby inflate COVID-19 mortality. The CDC (2021) notes that pediatric influenza deaths were virtually non-existent during the 2020–2021 season:



Peek (2021) reported in Scientific American earlier this year that “Flu Has Disappeared for More Than a Year” and enthusiastically attributed its precipitous decline to masks, social distancing, etc.



Credit: Katie Peek; Source: FluNet/Global Influenza Surveillance and Response System at the World Health Organization (data)

Later in this paper, I will detail why it does not make sense to attribute such a precipitous drop in influenza deaths to masks and social distancing, and why it seems *prima facie* more likely that deaths due to influenza and perhaps even deaths due to other human coronaviruses (HCoVs) are being systematically coded as deaths due to COVID-19. This is not to say that SARS-CoV-2 does not exist, or that it is somehow self-same with influenza. Rather, this point just underscores the importance of putting the official COVID-19 mortality figures in their proper context.

Additionally, when determining the effect of COVID-19 on overall mortality and thereby assessing its severity, it is important to consider trends in deaths from all causes, or, ‘all-cause mortality (ACM).’ Observing significant changes in ACM should indicate the degree to which COVID-19 affects overall mortality trends while at its seasonal peak. Consider the following data on Canadian ACM by week (ACM/w) over the last decade, presented by Rancourt et al. (2021):

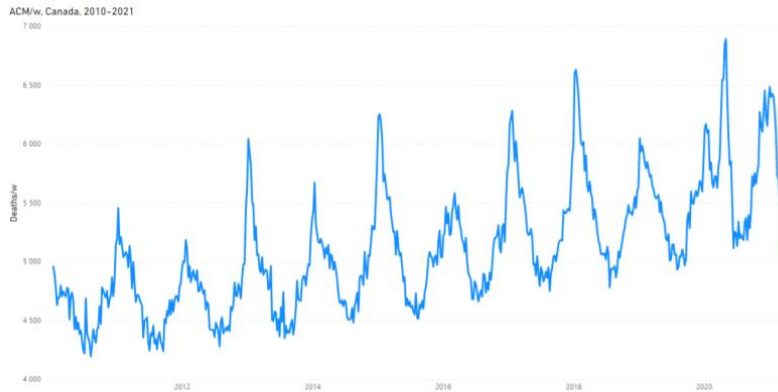


Figure 1a: All-cause mortality by week in Canada from 2010 to 2021. Data are displayed from January 2010 to March 2021. The y-scale is adjusted to show the region of interest. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

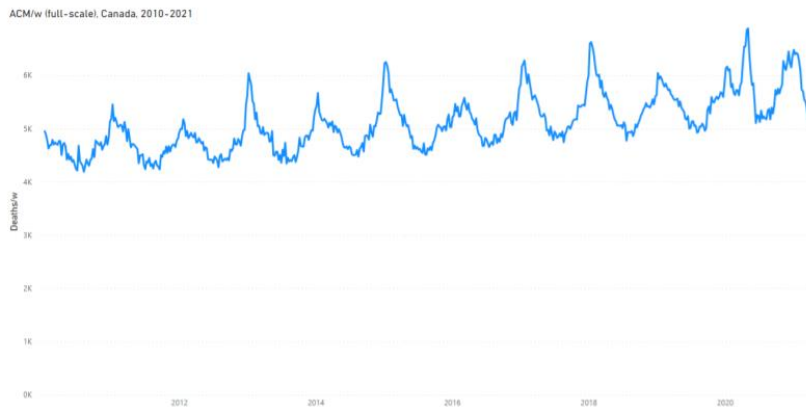


Figure 1b: All-cause mortality by week in Canada from 2010 to 2021. Data are displayed from January 2010 to March 2021. The y-scale is not adjusted to show only the region of interest; it starts from 0. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

As Rancourt et al. (2021) note, ACM/w for the 2019/2020 period peaked throughout the winter months (as it normally does) and *did not peak above the usual levels observed over the last decade*:

“Figure 1 shows that there was no excess yearly or seasonal mortality, above the usual values of the last decade for Canada, in either the 2019-2020 winter or the 2020-2021 winter (up to and including March 2021). This is confirmed by calculating ACM per year. We calculated ACM by “cycle-year”, where we define a cycle-year as occurring from week-31 (around the beginning of August) of calendar year N through to week-30 (around the end of July) of calendar year N+1. As such, for example, nominal cycle-year 2018 is centered on the winter of 2018-2019. This definition of cycle-year takes one from mid-summer-trough to the next mid-summer-trough in ACM/w, such as to capture the intrinsic seasonal structure of ACM/w, having winter highs and summer lows. The result is plotted in Figure 2:

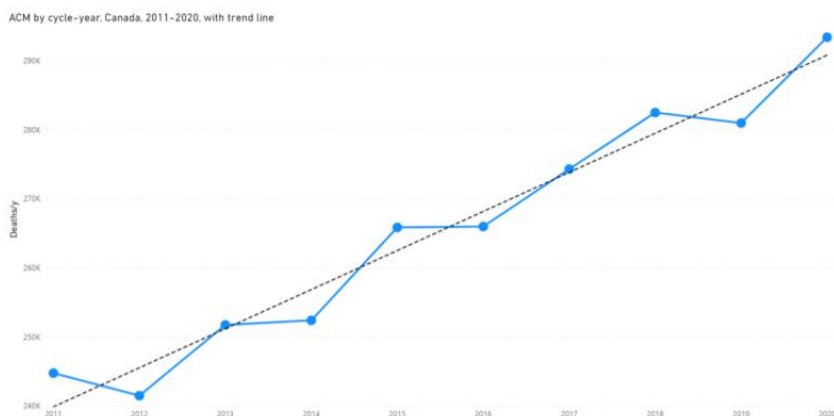
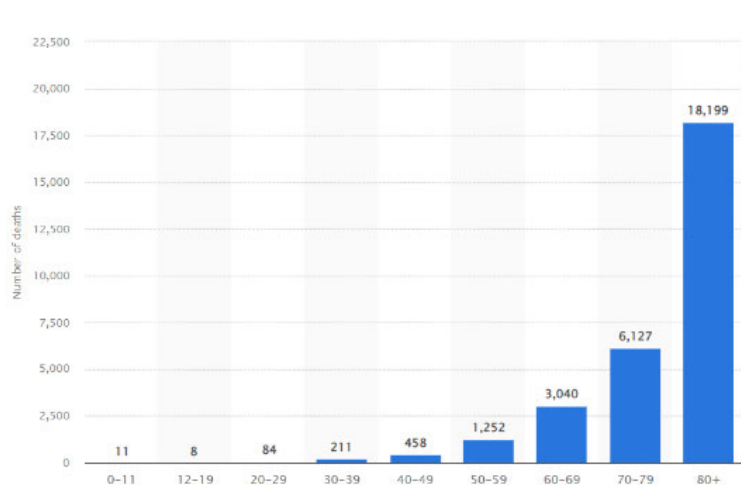


Figure 2: All-cause mortality by cycle-year for Canada, cycle-years 2011 to 2020, calculated as described above. The dashed line is a least-squares fitted straight line. The cycle-year starts on week-31 of a calendar year (beginning of August) and ends on week-30 of the next calendar year (end of July). Data for the calculation were retrieved from StatCan (StatCan, 2021), as described in section 2.

” (Rancourt, Baudin, and

Mercier, 2021)

Hence, after controlling for population growth, the overall number of people that one would expect to die in any given year, in Canada, did not significantly vary in 2020 when compared to other years over the last decade, and the same relationship holds for most other western countries as well (excepting the US). The fact that there are no anomalies in annual ACM mortality trends accords with the fact that as of November 26th, 2021, more than 60% of all COVID-19 decedents in Canada were aged 80+ years, which is near or beyond the average life expectancy of a Canadian citizen (Elflein, 2021). See the figure below:



(Elflein, 2021)

The same highly age-morbidity-specific COVID-19 mortality trend is observed in data from the United States and from literally every other country on Earth. See the following from Kostoff et al. (2021):

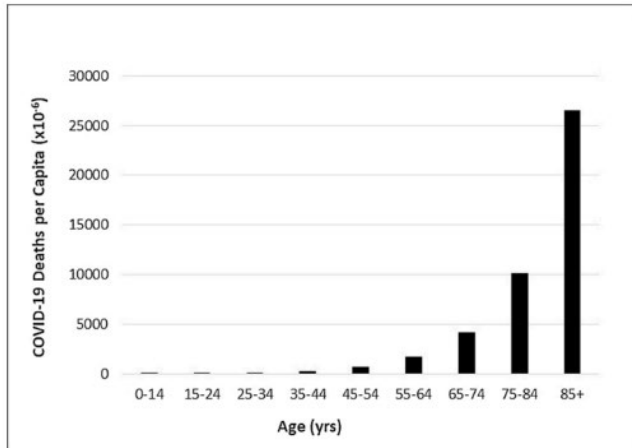


Fig. 1. COVID-19 Deaths per capita by age in the United States (as of Jun 5, 2021). Population-based on U.S. CDC WONDER Bridge-Race Population Estimate 2019. Data obtained from <https://wonder.cdc.gov/bridged-race-v2019.html> on 6/15/2021. Provisional COVID-19 deaths based on CDC data provided by the 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.

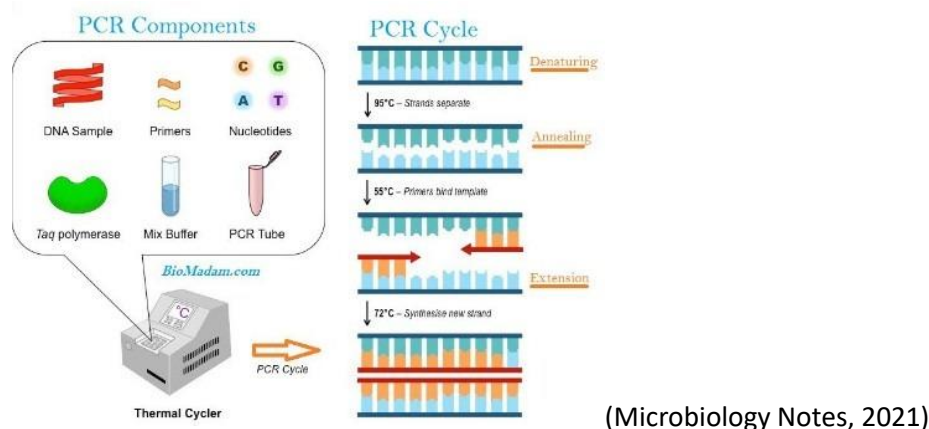
2. Mass Testing

Mass testing has played a significant role in the misclassification of COVID-19 hospitalizations and mortality. In analyzing data for case counts, hospitalizations, deaths, etc., we should keep in mind that the standards for confirming positive cases of SARS-CoV-2 infection via reverse transcription polymerase chain reaction (RT-PCR) testing, the so-called ‘gold standard’ of SARS-CoV-2 testing, are non-uniform from region to region. As Stang et al. (2021) note, “there is no international standardization across laboratories, rendering problematic the interpretation of RT-PCR tests when used as a tool for mass screening.” Typically, there are metrics that researchers can rely on to determine whether a positive RT-PCR test truly indicates the presence of an infectious case of SARS-CoV-2. However, if the tests are calibrated in such a way as to count non-infectious samples as infectious, then RT-PCR as a tool for mass screening will overestimate the number of infectious cases in a given population as well as the number of individuals in hospital for COVID-19 and not merely positive for mild or asymptomatic SARS-CoV-2 infection.

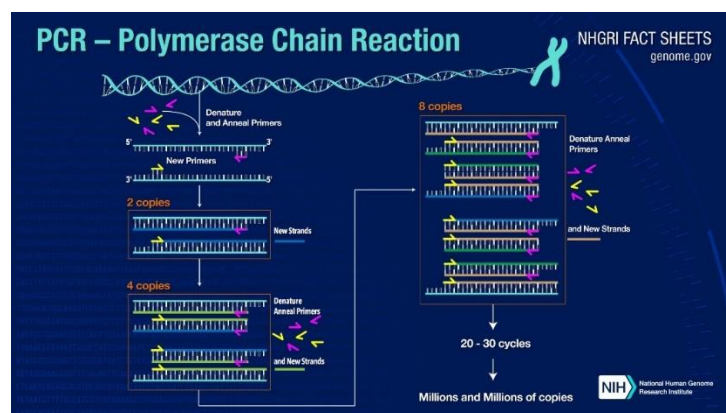
Even worse, if positive RT-PCR test results are observed in samples taken post-mortem or while patients are in hospital for reasons unrelated to COVID-19, then improper interpretation of the results will lead to inflated estimations of COVID-19 mortality. Testing of this sort was in fact carried out in countries like the US, UK, Italy, and Spain where it was “widely performed post-mortem on deceased patients. This resulted in falsely elevated case-fatality rates combined with massive underestimates of actual infections” (Reiss and Bhakdi, 2020). As I will show in this section, mass screening is indeed overestimating the number of truly infectious cases in many populations where ‘infectious’ just means ‘capable of passing SARS-CoV-2 onto another individual(s) such that said individual(s) is likely to (1) develop symptoms and/or (2) pass SARS-CoV-2 onto another individual(s).’ Most people who test positive for SARS-CoV-2 are non-infectious on this definition of infectious.

When detecting SARS-CoV-2 via RT-PCR, samples of presumed viral RNA are collected, typically via nasopharyngeal swab. First, the presumed viral RNA is synthesized into complementary DNA or ‘cDNA’

with an enzyme called reverse transcriptase (Microbiology Notes, 2021). Second, the cDNA is replicated or ‘amplified’ by a DNA polymerase enzyme (e.g., Taq polymerase) which initiates the polymerase chain reaction, i.e., a reaction catalyzed by DNA polymerase that doubles strands of cDNA, eventually resulting in millions of copies. Once the cDNA is sufficiently replicated, then its nucleotide sequences can be read and the type of genetic material presumed to be in the initial sample can be determined, e.g., viral SARS-CoV-2 RNA. Each time the cDNA is amplified, one ‘cycle’ passes. The number of cycles necessary to produce genetic material consistent with that of SARS-CoV-2’s genome is thought to correlate with the concentration of SARS-CoV-2 in the initial sample, i.e., the viral load (Stang et al., 2021). The number of cycles necessary to produce said genetic material is called the ‘cycle threshold (Ct) value.’ The Ct value is “inversely proportional to the initial amount of target nucleic acid and is thus a relative indicator of the concentration of viral particles in the clinical specimen” (Stang et al., 2021). Thus, so the logic goes, the lower the Ct value before genetic material indicating the presence of SARS-CoV-2’s genome is found, the higher the concentration of SARS-CoV-2 in the initial sample and the more likely it is that said sample harbours an *infectious* dose of SARS-CoV-2 and vice versa, given that viral load correlates with infectiousness (Stang et al., 2021). See the figures below for a visual representation of RT-PCR testing:



(Microbiology Notes, 2021)



(NIH 2021)

Importantly, researchers must set and adhere to a *maximum* Ct value when conducting RT-PCR tests so as to not amplify the DNA past the point where meaningful conclusions can be drawn. RT-PCR is so sensitive that “evaluation of transcript from a single cell is possible” (Microbiology Noes, 2021). Indeed, the late inventor of the RT-PCR method and esteemed Nobel laureate, Kary Mullis, has been posthumously quoted during the COVID-era as describing how the results of RT-PCR tests can be easily misinterpreted if the tests are not properly parameterized and calibrated for the specific type of genetic

material under examination. Mullis, while discussing the utility of RT-PCR for diagnosing HIV/AIDS, notes that “with PCR, if you do it well, you can find almost anything in anybody. It starts making you believe in the sort of Buddhist notion that everything is contained in everything else ... Because, *if you can amplify one single molecule up to something that you can really measure, which PCR can do, then there are just very few molecules for which you don’t have at least a single one of them in your body* ... It [PCR] allows you to take a very miniscule amount of anything and make it measurable and then talk about it in meetings and stuff like it is important. See, that’s not a misuse, that’s just sort of a misinterpretation [my emphasis]” (J Wilderness, 2020).

One of the ways in which the results of RT-PCR tests can be misinterpreted in the sense that Mullis meant is if maximum Ct values are set too high. If maximum Ct values for determining infectious cases of SARS-CoV-2 infection are set too high, then researchers risk classifying non-infectious individuals as infectious, or, in some cases, generating false positives and thereby inflating the number of presumed infectious individuals in a population. Consider the following from Stang et al. (2021) (i.e., ‘no’ means no symptoms, ‘yes’ means yes symptoms):

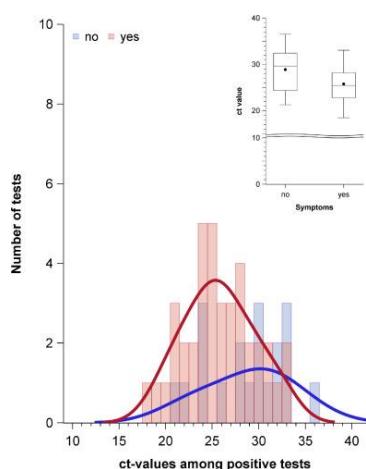


Figure 1 Ct value distribution among symptomatic and asymptomatic individuals’ with positive tests in the region of Münster, North Rhine-Westphalia, Germany, 2020

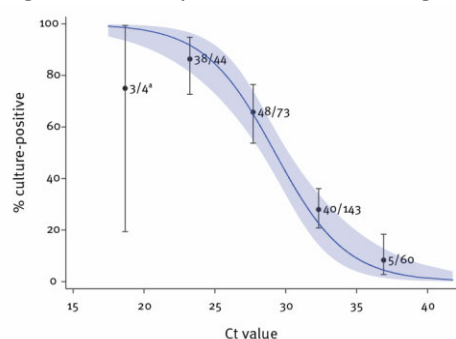
As we can see, relatively few symptomatic individuals cluster along the low-end of observed Ct values. Most test positive at higher Ct values, indicating that asymptomatic individuals have low viral loads and are, therefore, (in most cases) non-infectious. The fact that most asymptomatic people are non-infectious should prompt us to question why RT-PCR would ever be used to mass screen asymptomatic populations and generate data that inform public policy on lockdowns, masks, and so forth. Stang et al. (2021) observed the following in their study of the results of 80% of all RT-PCR tests performed during the peaks of the first and second waves in Münster, Germany:

“In light of our findings that more than half of individuals with positive PCR test results are unlikely to have been infectious, RT-PCR test positivity should not be taken as an accurate measure of infectious SARS-CoV-2 incidence. Our results confirm the findings of others that the routine use of “positive” RT-PCR test results as the gold standard for assessing and controlling infectiousness fails to reflect the fact “that 50-75% of the time an individual is PCR positive, they are likely to be post-infectious [sic].

Asymptomatic individuals with positive RT-PCR test results have higher Ct values and a lower probability of being infectious than symptomatic individuals with positive results. Although Ct values have been shown to be inversely associated with viral load and infectivity, there is no international standardization across laboratories, rendering problematic the interpretation of RT-PCR tests when used as a tool for mass screening.”

The relationship between Ct values and infectiousness is well-characterized. In their study of RT-PCR positive test results in England, Singanayagam et al. (2020) “observed a strong relationship between Ct value and ability to recover infectious virus [from a sample].” The researchers were able to recover infectious virus far more often from samples that tested positive with lower Ct values than from samples that tested positive with higher Ct values (Singanayagam et al., 2020). They note that “Virus propagation was successful from five [8.33%] of 60 samples with Ct > 35; all five were from symptomatic cases and none had severe illness.” See the following figures:

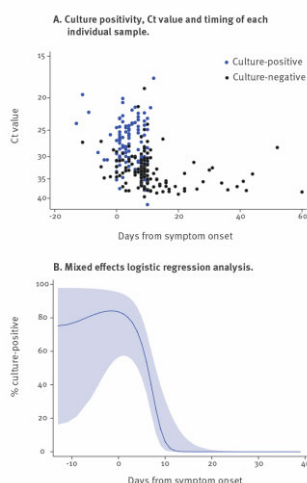
“Figure 2. Relationship between RT-PCR Ct value and culture positivity in mixed effects logistic regression analysis, SARS-CoV-2, England, January–May 2020 (n = 324)



Ct: cycle threshold.

^a Wide confidence intervals due to small sample size”. (Singanayagam et al., 2020).

Figure 3. Relationship between culture positivity and time between symptom onset and sample collection, SARS-CoV-2, England, January–May 2020 (n = 246)



Ct = cycle threshold. The y axis on Figure 3A was created in Stata 15.1 with reversed log scale*.” (Singanayagam et al., 2020).

As we can see, the likelihood that any infectious genetic material whatsoever is present in a sample drops precipitously for samples that test positive after 20–25 cycles. This is extremely troubling given that most governments in western countries currently recognize samples as infectious even if they test positive with Ct values that greatly exceed 20–25. For instance, Public Health Ontario (PHO) adheres to the following testing guidelines with respect to test-positivity and Ct values:

“At PHO, we have developed a PCR test in our lab, with positive and negative cutoff points. The cutoff point for a positive result for PHO’s developed lab test is 38 cycles. This means that if the virus is found at or before 38 cycles are completed, then the test is considered positive. The cutoff point for a negative result is 40 cycles. If the virus is detected between 38 and 40 cycles, we call this an indeterminate or inconclusive result. All inconclusive results are considered probable (likely) cases for public health reporting.” (Public Health Ontario, 2020).

Given that infectious virus cannot be cultured from samples that test positive at Ct values > 35, PHO’s maximum Ct value is currently set far too high and will therefore overestimate the number of infectious individuals in the general population (Singanayagam et al., 2020). These policies are leading to the quarantining of non-infectious individuals and the coding of hospitalizations and deaths as ‘COVID-19 hospitalizations and/or deaths’ when there is, in fact, *no infectious disease present*. Cevik et al. (2021) note that the duration of viable viral shedding (i.e., the duration of time in which infected individuals are capable of passing viable virus onto other individuals) is such that the mere presence of genetic material consistent with a particular viral genome “cannot be used to infer infectiousness.” It could be, as Cevik et al. (2021) note, that the majority of asymptomatic individuals who test positive (even at lower Ct values) are *post-infectious*. Through their analysis of 79 studies on SARS-CoV-2, 8 on SARS-CoV, and 11 on MERS-CoV, Cevik et al. (2021) confirmed that non-viable viral shedding can occur for a maximum of “83 days in the upper respiratory tract, 59 days in the lower respiratory tract, 126 days in stools, and 60 days in serum.” Thus, individuals can test ‘positive’ for SARS-CoV-2 infection for at least 83 days onset initial infection, despite being (1) totally non-infectious, and (2) not at all symptomatic.

The duration of shedding for *viable* infectious virus, in comparison, is relatively short-lived, i.e., “No study detected live virus beyond day 9 of illness, despite persistently high viral loads, which were inferred from cycle threshold values” (Cevik et al. 2021). Similarly, Walsh et al. (2020) found that “In symptomatic patients, there is evidence of a reduction in infectivity 7–10 days after onset of symptoms.” Scola and Bernard et al. (2020) found that “Correlation between successful isolation of virus in cell culture and Ct value of quantitative RT-PCR targeting E-gene suggests that patients with Ct above 33-34 are not contagious and thus can be discharged from hospital care.” Jaafar et al. (2020) observed that “at Ct = 25, up to 70% of patients remain positive in culture and that at Ct = 30 this value drops to 20% ... At Ct = 35, the value we used to report a positive result for PCR, <3% of cultures are positive.” Boretti (2020) even found that “There was no growth in samples with a Ct > 24 or STT [symptom onset to test] > 8 days.” In light of these data, it seems that mass screening for SARS-CoV-2 via RT-PCR testing (the so-called ‘gold standard’ of testing for SARS-CoV-2) is an extremely poor way to determine both the number of truly infectious individuals active in the community at any given time and the true burden on hospitals due to COVID-19. It also seems non-sensical to subject asymptomatic individuals to mass testing (even in hospital) given the strong correlation between *symptomatic* illness, viral load (represented as Ct value), and infectiousness.

3. Objection: Would COVID-19 Have Been Far More Deadly Had Governments Not Instituted Restrictive Non-Pharmaceutical Interventions (e.g., Lockdowns)? Did Our Governments Successfully Prevent a Public Health Disaster?

One might retort that the only reason why IFRs and overall mortality in most western countries have been so low is because strict non-pharmaceutical interventions (NPIs) work extraordinarily well at arresting epidemiologically relevant transmission, reducing hospitalizations, mortality, etc. Likewise, one might argue that widespread adherence to masking and social distancing policies kept the IFR, reproduction number (R_0), etc., of SARS-CoV-2 well below what they would have been if no such measures were taken. Hence, if we did not take these measures, then SARS-CoV-2 would have been much more deadly than it turned out to be, or so the argument goes. This is an absurd argument for a number of reasons, notwithstanding the fact that we now have evidence to suggest that strict NPIs, universal masking, etc., do not correlate with significantly arrested transmission of aerosolized respiratory viruses. I will detail this evidence shortly, but for now, recall that even the ‘worst-case scenario’ predictions that *did* account for the introduction of NPIs from back in March, 2020, *all failed miserably to materialize*. A better explanation for lower-than-anticipated mortality is that the initial predictive models were just extremely flawed in both their assumptions and the data they relied on to make their predictions.

Indeed, back in March, 2020, CNN reported on predictive modeling conducted by a team of researchers from Imperial College London (ICL), UK (Walsh, 2021). Their modelling was highly influential inasmuch as it provided the initial justification for governments to lockdown their populations on the assumption that there would occur at least 250,000 deaths in the UK and 1.1–1.2 million in the US *with lockdowns* and *with treatments available* within just a few months of the initial outbreaks in Wuhan (Ferguson et al., 2020). The ‘worst-case scenario’ predictions for the US and UK respectively, the researchers reported, forecasted that 2.2 million and 510,000 would die if governments did nothing at all, and these figures did not include additional mortality from the possibility that healthcare systems would be overwhelmed (Ferguson et al., 2020). There *were* lockdowns in nearly every western country, but there was very little in the way of treatment (for the most part, people merely sheltered in place), and still, the ‘best-case scenario’ predictions did not even come close to materializing anywhere in the world. Hence, the ICL models vastly overestimated the ability of SARS-CoV-2 to cause death. Consider the following figure mapping the ICL researchers’ predicted pandemic scenarios for between April and August, 2020:

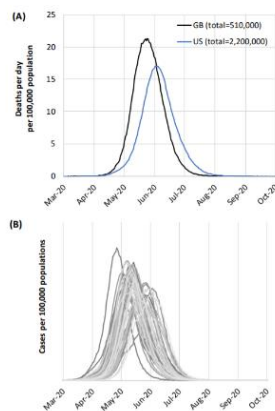


Figure 1: Unmitigated epidemic scenarios for GB and the US. (A) Projected deaths per day per 100,000 population in GB and US. (B) Case epidemic trajectories across the US by state.

(Ferguson et al., 2020)

Needless to say, the ICL models and others like them have been critiqued into oblivion, and for good reason. For instance, the researchers made unfounded assumptions about the IFR and R0 of SARS-CoV-2 that were inferred primarily from extremely limited data gathered from the intensive care units (ICUs) of hospitals in China and Italy (Ioannidis, 2020). Hence, the models suffered from perhaps the most extreme form of selection bias in the history of public health. Indeed, eminent epidemiologists were vocal with their criticisms from the start. They warned against instituting blanket lockdowns based on flawed assumptions, but, their advice fell on deaf ears. Consider the following, written by Ioannidis (2020) and published March 17th, 2020:

“The data collected so far on how many people are infected and how the epidemic is evolving are utterly unreliable. Given the limited testing to date, some deaths and probably the vast majority of infections due to SARS-CoV-2 are being missed. We don’t know if we are failing to capture infections by a factor of three or 300. Three months after the outbreak emerged, most countries, including the U.S., lack the ability to test a large number of people and no countries have reliable data on the prevalence of the virus in a representative random sample of the general population.

...

This evidence fiasco creates tremendous uncertainty about the risk of dying from Covid-19. Reported case fatality rates, like the official 3.4% rate from the World Health Organization, cause horror – and are meaningless. *Patients who have been tested for SARS-CoV-2 are disproportionately those with severe symptoms and bad outcomes.* As most health systems have limited testing capacity, *selection bias may even worsen in the near future.*

...

Projecting the Diamond Princess mortality rate onto the age structure of the U.S. population, the death rate among people infected with Covid-19 would be 0.125%. But since this estimate is based on extremely thin data – there were just seven deaths among the 700 infected passengers and crew – the real death rate could stretch from five times lower (0.025%) to five times higher (0.625%). It is also possible that some of the passengers who were infected might die later, and that tourists may have different frequencies of chronic diseases — a risk factor for worse outcomes with SARS-CoV-2 infection — than the general population. Adding these extra sources of uncertainty, reasonable estimates for the case fatality ratio in the general U.S. population vary from 0.05% to 1%.”

Ioannidis’ critiques were borne out by subsequent, better quality data showing median IFRs as low as 0.59% for individuals under aged 60–69, and even as low as 0.0027% for people aged 0–19 (Axfors and Ioannidis 2021). Absolute mortality risks, as we now know, can be as low as 0.0002% for people under 18 years of age, and infections are far more widespread than was initially estimated (Ledford, 2021; Ioannidis, 2021). Media sensationalism based on poor quality data and highly influential predictive modelling from ICL that *relied* on these poor quality data informed the extreme, disproportionate, draconian measures (i.e., lockdowns, surveillance, masking, etc.) that followed the panic. Indeed, the influence of the ICL modelling on public officials cannot be overstated. Observe:



(Bostock, 2020)

In addition to relying on poor quality data, the ICL researchers made a few glaringly false assumptions. For instance, they assumed that since SARS-CoV-2 was a ‘novel’ coronavirus, no one would have any appreciable degree of pre-existing immunity. Consider the following from Adam (2020):

“The simplest SIR [susceptible, infected, recovered] models make basic assumptions, such as that *everyone has the same chance of catching the virus from an infected person because the population is perfectly and evenly mixed, and that people with the disease are all equally infectious until they die or recover*. More-advanced models, which make the quantitative predictions policymakers need during an emerging pandemic, subdivide people into smaller groups – by age, sex, health status, employment, number of contacts, and so on – to set who meets whom, when and in which places (see ‘Measuring social mixing’).

...

Some parameters, meanwhile, must be entirely assumed. *The Imperial team had to surmise, for instance, that there is no natural immunity to COVID-19 – so the entire population starts out in the susceptible group – and that people who recover from COVID-19 are immune to reinfection in the short term [my emphasis].*”

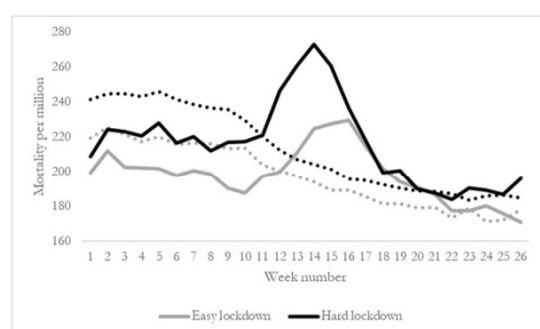
These assumptions cannot be ignored. They are so extraordinarily false and absurd in hindsight given current and longstanding research on the nature of naturally acquired immunity to betacoronaviruses. In the next section, I will detail the prevalence of naturally acquired and/or pre-existing immunity to SARS-CoV-2. In short, there was never any reason to assume that zero percent of people would have protective immunity against SARS-CoV-2 given its marked genetic similarities to other common betacoronaviruses that circulate constantly in human populations. These coronaviruses can generate protective cross-reactive immunity against SARS-CoV-2 and its variants, thereby significantly reducing the number of susceptible people and thereby reducing the disease burden.

Still, one might be tempted to claim that NPIs such as masks, lockdowns, and prolonged social distancing measures had an appreciable effect on COVID-19 mortality and infections. They did not. Bjørnskov (2021), for instance, surveyed transmission data from northern Jutland, Denmark and found that “while infection levels decreased, they did so *before* lockdown was effective, and infection numbers also decreased in neighbour municipalities without mandates.” Bjørnskov (2021) also concludes that “direct spill-over to neighbour municipalities or the simultaneous mass testing do not explain this ... data suggest that efficient infection surveillance and voluntary compliance *make full lockdowns unnecessary.*” Likewise, Bjørnskov (2021) found the following in a broader survey of western countries:

“The lockdowns in most Western countries have thrown the world into the most severe recession since World War II and the most rapidly developing recession ever seen in mature market economies. They have also caused an erosion of fundamental rights and the separation of powers in a large part of the world as both democratic and autocratic regimes have misused their emergency powers and ignored constitutional limits to policy-making [sic]. It is therefore important to evaluate whether and to which extent the lockdowns have worked as officially intended: to suppress the spread of the SARS-CoV-2 virus and prevent deaths associated with it. *Comparing weekly mortality in 24 European countries, the findings in this paper suggest that more severe lockdown policies have not been associated with lower mortality.* In other words, the lockdowns have not worked as intended [my emphasis].”

See the following from Bjørnskov (2021):

Figure 1. Mortality development in 2020, two groups



Wood (2020) found that “A Bayesian inverse problem approach applied to UK data on first wave Covid-19 deaths and the disease duration distribution suggests that fatal infections were in decline before full UK lockdown (24 March 2020), and that fatal infections in Sweden started to decline only a day or two later.” In the top left curve of the figure below, notice that COVID-19 mortality peaked and began to decline for multiple weeks before lockdowns went into effect:

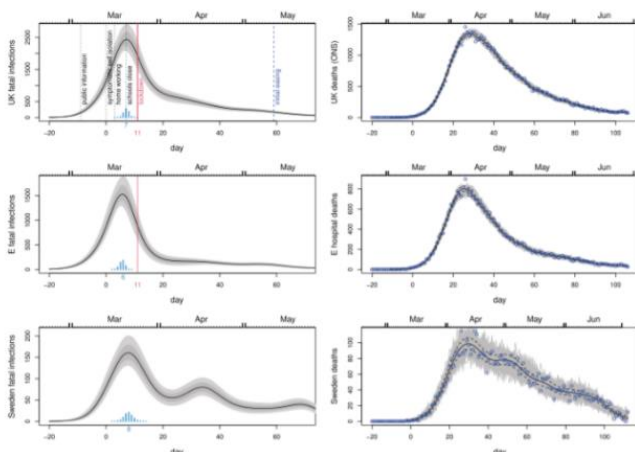


FIGURE 2 In all plots, black curves show the posterior median, while light grey and dark grey regions show, respectively, 95% and 68% confidence regions, including uncertainty in the fatal disease duration distribution. Day 0 is 13th March 2020, and the vertical red line marks the first day of UK lockdown. Top left: Inferred daily fatal infection rate, f_{fat} , for the United Kingdom. The scaled bar chart shows the posterior distribution for day of peak infection with the peak day labelled. NPI start dates are marked by labelled vertical lines. Top right: Consistency check. In grey are 100 sets of death data simulated forward from the inferred median fatal infection profile. Symbols are the ONS daily death data for the United Kingdom on which inference is based. The dashed curves are 95% confidence intervals for underlying death rate estimated by direct fitting of (1). Middle row: As top row, but using the NHS England daily hospital death data. Note that the inferred infection trajectories are substantially different from time-lagged versions of the deaths trajectories. Bottom row: as the previous rows, but for Sweden. This figure appears in colour in the electronic version of this article, and any mention of colour refers to that version

(Wood, 2020)

Hunter et al. (2021) produced similar findings. They note that “stay at home orders and closure of all non-businesses was not associated with any independent additional impact” (Hunter et al., 2021). Berry et al. (2021) argue that “shelter-in-place orders had no detectable health benefits, only modest effects on behavior, and small but adverse effects on the economy” (Berry et al., 2021). Importantly, we have control groups, i.e., countries that did not impose strict NPIs. Consider the following from Karáth (2020):

“Belarus’s beleaguered government remains unfazed by covid-19. President Aleksander Lukashenko, who has been in power since 1994, has flatly denied the seriousness of the pandemic, refusing to impose a lockdown, close schools, or cancel mass events like the Belarusian football league or the Victory Day parade. Yet the country’s death rate is among the lowest in Europe – just over 700 in a population of 9.5 million with over 73,000 confirmed cases.”

Extrapolating from pre-lockdown growth rate trends in COVID-19 mortality, Meunier (2020) found that lockdowns “might not have saved any life in western Europe.” For instance, “neighboring countries applying less restrictive social distancing measures [e.g., Sweden, Belarus] (as opposed to police-enforced home containment) experience a very similar time evolution of the epidemic” (Meunier, 2020). In sum, Meunier (2020) concludes the following:

“Our results show a general decay trend in the growth rates and reproduction numbers two to three weeks before the full lockdown policies would be expected to have visible effects. Comparison of pre and post lockdown observations reveals a counter-intuitive slowdown in the decay of the epidemic after lockdown. *Estimates of daily and total deaths numbers using pre-lockdown trends suggest that no lives were saved by this strategy*, in comparison with pre-lockdown, less restrictive, social distancing policies [my emphasis].”

Gibson (2020) found that “Lockdowns do not reduce Covid-19 deaths.” In New Zealand, for instance, the “ineffectiveness of lockdowns suggests that New Zealand suffered large economic costs for little benefit in terms of lives saved” (Gibson, 2020). For the United States, “There is no evidence that counties with a lockdown have fewer deaths ... For all three dates, the coefficient on lockdown is statistically insignificant” (Gibson, 2020). Crucially, through an analysis of NPI implementation in subnational regions of 10 countries: England, France, Germany, Iran, Italy, Netherlands, Spain, South Korea, Sweden and the United States, Bendavid et al. (2021) found the following:

“After subtracting the epidemic and I_r NPI [less-restrictive non-pharmaceutical interventions] effects, *we find no clear, significant beneficial effect of m rNPIs [more-restrictive non-pharmaceutical interventions] on case growth in any country*. In France, for example, the effect of m rNPIs was +7% (95% CI: -5%-19%) when compared with Sweden and + 13% (-12%-38%) when compared with South Korea (positive means pro-contagion). The 95% confidence intervals excluded 30% declines in all 16 comparisons and 15% declines in 11/16 comparisons.

...

While small benefits cannot be excluded, we do not find significant benefits on case growth of more restrictive NPIs. Similar reductions in case growth may be achievable with less-restrictive interventions.”

Similar results have been replicated *ad infimum*. Chaudhry et al. (2021) found that “Rapid border closures, full lockdowns, and wide-spread testing were not associated with COVID-19 mortality per million people.” The cost-benefit ratio of lockdowns, clearly, was never seriously considered by the ICL modellers. They only very myopically considered the costs and benefits of *potentially* reducing deaths from COVID-19. They flatly ignored the obvious socioeconomic fallout that lockdowns generate, as well as the mental health toll and strain on medical care as a result of missed surgeries, etc. Since March, 2020, many cost-benefit analyses have been conducted, but only after the damage was already done, unfortunately. They almost universally find that lockdowns cause more harm than they prevent. Consider this from Halperin et al. (2021):

“The cumulative evidence suggests that “sledge-hammer” lockdown approaches, such as the closing of all non-essential workplaces and schools, should be avoided in favor of more effective, carefully targeted “scalpel” public health strategies [sic]. Indiscriminate lockdowns have had far-reaching unintended consequences, disproportionately affecting socioeconomically disadvantaged and vulnerable populations. Other consequences include alarming increases in mental health problems (e.g., depression, anxiety, and social isolation), drug overdose, domestic violence, child abuse, weight gain, abuse by law enforcement in some places, and discontinuation of non-COVID-19 clinical services and prevention programs [sic].”

The cumulative effects of these harms are such that some economists estimate that the quality-adjusted life years (QALYs) lost because of lockdowns *exceed those that would have been lost, even in the dreaded ‘worst-case scenario’ predictions, because of COVID-19!* Consider these results from Price (2021) suggesting that QALYs lost from missed cancer diagnoses in the UK might exceed those lost because of deaths directly related to COVID-19:

In just four cancer types (breast, colon, lung and oesophagus), studies during the first wave of the COVID-19 pandemic (published July 2020 [sic]) predicted 60,000 lost life years. The quality-adjusted life years and the productivity losses due to these excess cancer deaths have been estimated in this new article to be 32,700 and £104 million over 5 years, respectively. This is nearly 1.5 times higher per capita than that of deaths directly related to COVID-19 in that time. The authors confirm that this is a conservative estimate for these cancer groups as it does not take into account additional productivity losses due to delays or reduction in quality of treatment and stage migration.”

Likewise, Maringe et al. (2021) modelled the impact of UK lockdown policies on cancer-related deaths from misdiagnoses and delayed surgeries. They found that “The total additional YLLs across these cancers is estimated to be 59,204–63,229 years” (Maringe et al., 2021). Ultimately, “Substantial increases in the number of avoidable cancer deaths in England are to be expected as a result of diagnostic delays due to the COVID-19 pandemic in the UK” (Maringe et al., 2021). Lally (2021) estimated QALYs lost due to lockdowns in New Zealand and found the following:

“Using data available up to 28 June 2021, the estimated additional deaths from a mitigation strategy are 1,750 to 4,600, implying a Cost per Quality Adjusted Life Year saved by locking down in March 2020 of at least 13 times the generally employed threshold figure of \$62,000 for health interventions in New Zealand; the lockdowns do not then seem to have been justified by reference to the standard benchmark. Using only data available to the New Zealand government

in March 2020, the ratio is similar and therefore the same conclusion holds that the nation-wide lockdown strategy was not warranted.”

In sum, lockdowns were an abysmal failure of public policy in every respect. One might still argue that masks had some appreciable effect on COVID-19 mortality and/or transmission. I have a difficult time taking these claims seriously enough to address them, but I will present evidence from nearly every randomized control trial (RCT) to date showing that facemasks have no appreciable effect on the transmission of aerosolized respiratory particles and, therefore, on viral transmission. Bundgaard et al. (2020) found that “The recommendation to wear surgical masks to supplement other public health measures did not reduce the SARS-CoV-2 infection rate among wearers by more than 50% in a community with modest infection rates, some degree of social distancing, and uncommon general mask use.” Unsurprisingly, their results were wrongfully maligned in the media and by public health officials:

“DANMASK-19, the first trial of mask use during covid-19, was “negative.” Masks didn’t work. We knew this before the trial was published because we were told so on social media. The authors were reported by the media to be struggling to find a major journal for their trial. Journals weren’t proving brave enough to publish the study, said the authors, and they didn’t make a preprint available.” (Abbasi, 2020).

Hence, from the start, the inane masking debate has been subject to intense *political* (not scientific) disagreement. To date, no good quality evidence suggests that they work. Prior evidence of their ineffectiveness was systematically ignored by public health officials, and the sheer absence of evidence was even publicly *acknowledged* in some instances by organizations like the World Health Organization (WHO). Indeed, early on, Klompas et al. (2020) noted the following in an article published in the *New England Journal of Medicine (NEJM)*:

“We know that wearing a mask outside health care facilities offers little, if any, protection from infection. Public health authorities define a significant exposure to Covid-19 as face-to-face contact within 6 feet with a patient with symptomatic Covid-19 that is sustained for at least a few minutes (and some say more than 10 minutes or even 30 minutes). The chance of catching Covid-19 from a passing interaction in a public space is therefore minimal. In many cases, the desire for widespread masking is a reflexive reaction to anxiety over the pandemic.”

The quote above is delivered by its author with a tone of obviousness, and for good reason. The researchers were likely aware of the large amount of good quality data suggesting that masks do not work to prevent viral transmission in both community and healthcare settings. For instance, in an RCT that examined the effects of cloth and medical masks for reducing clinical respiratory illness (CRI), influenza-like illness (ILI) and laboratory-confirmed respiratory virus infection, MacIntyre et al. (2015) concluded that their results “caution against the use of cloth masks.” This is because, as they note, “Moisture retention, reuse of cloth masks and poor filtration may result in *increased risk of infection* [my emphasis]” (MacIntyre et al., 2015). While studying the transmission of common-cold-causing coronaviruses in Japan, Jacobs et al. (2009) found that “Face mask use in health care workers has not been demonstrated to provide benefit in terms of cold symptoms or getting colds.” Likewise, in a trial meant to study the effectiveness of cloth masks for preventing influenza transmission, Cowling et al. (2010) found that “There is little evidence to support the effectiveness of face masks to reduce the risk of infection.” Bin-Reza et al. (2012) conducted a meta-analysis of 17 studies on the use of cloth masks and found that “None of the studies established a conclusive relationship between mask/respirator use

and protection against influenza infection.” Smith et al. (2016) reviewed 6 clinical studies and observed “no significant difference between N95 respirators and surgical masks in associated risk of (a) laboratory-confirmed respiratory infection, (b) influenza-like illness, or (c) reported work-place absenteeism.” Similarly, Radonovich et al. (2019) found that “Among 2862 randomized participants, 2371 completed the study and accounted for 5180 HCW-seasons. ... Among outpatient health care personnel, N95 respirators vs medical masks as worn by participants in this trial resulted in no significant difference in the incidence of laboratory-confirmed influenza.”

Many of the observational studies on masking conducted during the COVID-era are highly prone to bias and confounding variables, such as enhanced caution, seasonality regarding the spread of aerosolized respiratory viruses, etc. Consider this from Dugré et al. (2020):

“In observational studies, wearing masks is associated with a lower risk of contracting viral respiratory infections. For example, in a 2011 Cochrane systematic review published by Jefferson et al, the use of masks in case-control studies was associated with an important risk reduction (odds ratio = 0.32; 95% CI 0.26 to 0.39). However, the observational design is at high risk of confounding and mask use might simply be a surrogate measure for comparing more careful versus less careful people. Experimental laboratory-based studies of masks and mask types seem to provide promising and important information, but translation into meaningful clinical differences is often lacking.”

Desai and Mehrotra (2020) concluded that “Face masks should not be worn by healthy individuals to protect themselves from acquiring respiratory infection because there is no evidence to suggest that face masks worn by healthy individuals are effective in preventing people from becoming ill.” Peebles (2021) recently reported on a large RCT conducted by Abaluck et al. (2021) in Bangladesh suggesting that masking may have *some* effect on reducing viral transmission. However, the study is very flawed. Consider the following from Rancourt (2021):

“The cluster-randomized trial study of Abaluck *et al.* (2021) is fatally flawed, and therefore of no value for informing public health policy, for two main reasons:

1. The antibody detection was performed using a single commercial FDA emergency-use-authorized (EUA) serology test that is not suitable for the intended application to SARS-CoV-2 in Bangladesh (not calibrated or validated for populations in Bangladesh; undetermined cross-reactivity against broad-array IgM antibodies, malaria, influenza, etc.).
2. The participants (individual level, family level, village level) in the control and treatment arms were systematically handled in palpably different ways that are linked to factors established to be strongly associated to infection and severity with viral respiratory diseases, in particular, and to individual health in general.

These disjunctive fatal flaws are explained below. Either one is sufficient to invalidate the results and conclusions.”

Better quality systematic reviews and meta-analyses come to more reliable conclusions. For instance, in a review conducted by Lipp and Edwards (2014) involving a total of 2113 participants, they observed “no statistically significant difference in infection rates between the masked and unmasked group in any of the trials.” In their review, Jefferson et al. (2020) found the following:

“Most included trials had poor design, reporting and sparse events. There was insufficient evidence to provide a recommendation on the use of facial barriers without other measures. We found insufficient evidence for a difference between surgical masks and N95 respirators and limited evidence to support effectiveness of quarantine. Based on observational evidence from the previous SARS epidemic included in the previous version of our Cochrane review we recommend the use of masks combined with other measures.”

In sum, the relatively low IFR and overall deadliness of SARS-CoV-2 infection have almost nothing to do with strict lockdowns, masking, etc. They have more to do with the intrinsic properties of SARS-CoV-2 itself, and the overall health status of the populations in which it circulates. The most influential models that predicted COVID-19 mortality were extremely flawed from the outset, and they stirred excessive panic for no good reason, at least from a public health-based perspective. Hence, COVID-19 is not particularly deadly for most people, and our elected officials *did not* prevent a public health disaster by instituting harsh measures.

Premise 2: Naturally acquired immunity against infection by SARS-CoV-2 is widespread and highly protective

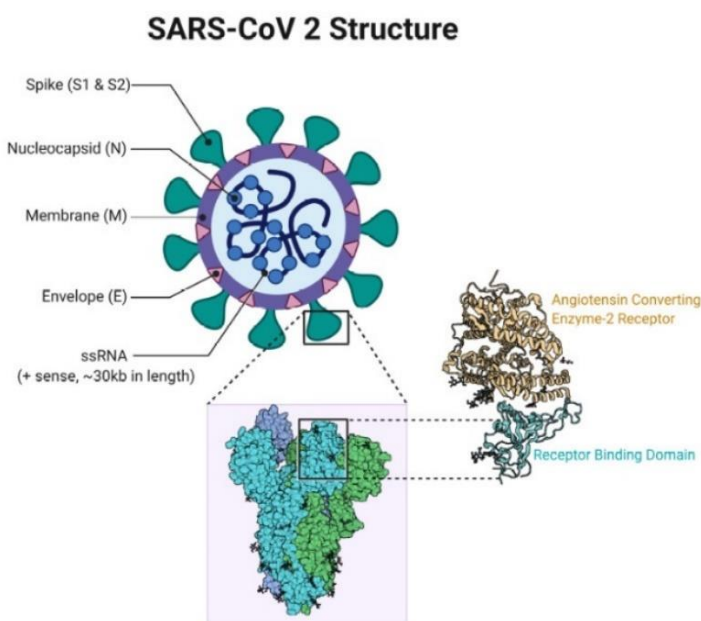
Evidently, one should advocate for mass vaccination against a pathogen only if (1) the pathogen is particularly deadly, and (2) naturally acquired immunity is not already widespread. Already, I have shown that (1) is false in the case of SARS-CoV-2. On the other hand, if relatively few people have been exposed, it might still be worth it to mass-vaccinate, so long as the vaccines in question are safe and effective. In this section, I will provide evidence to suggest that naturally acquired immunity against SARS-CoV-2 is widespread and that it is on par with or even better in terms of preventing symptomatic infection/reinfection than vaccine-induced immunity. It follows, therefore, that mass vaccination is totally unjustified.

Using national databases comprising more than 350,000 persons tested for SARS-CoV-2 via RT-PCR, Abu-Raddad et al. (2021) found that “There were no cases of critical disease at reinfection and 28 cases at primary infection (Table S3), for an odds ratio of 0.00 (95% CI, 0.00 to 0.64).” Their findings point to the following explanation: “Reinfections had 90% lower odds of resulting in hospitalization or death than primary infections. Four reinfections were severe enough to lead to acute care hospitalization. None led to hospitalization in an ICU, and none ended in death. Reinfections were rare and were generally mild, *perhaps because of the primed immune system after primary infection* [my emphasis].” (Abu-Raddad et al., 2021). Critics of acknowledging natural immunity as a valid form of protection might suggest that it is unethical to allow anyone to be exposed to SARS-CoV-2, and so we should always prefer vaccination over natural infection for conferring immunity. In the next section, I will show that this argument rests on two flawed premises. First, it assumes that the currently available vaccines are especially effective at preventing infection for long periods of time *without posing serious risks of their own* (which is false). Secondly, this argument implies that mild to asymptomatic infections are incapable of conferring protective immunity against SARS-CoV-2. As I will show in this section, this is not the case. Mild or asymptomatic infection by SARS-CoV-2 (which clearly portends no or exceedingly few negative health outcomes for the infected) is *sufficient to confer long-lasting protective natural immunity against SARS-CoV-2*.

Gazit et al. (2021) conducted “a retrospective observational study comparing three groups: (1) SARS-CoV-2-naïve individuals [i.e., not previously infected] who received a two-dose regimen of the

BioNTech/Pfizer mRNA BNT162b2 vaccine, (2) previously infected individuals who have not been vaccinated, and (3) previously infected *and* single dose vaccinated individuals.” Their results suggest that “SARS-CoV-2-naïve vaccinees had a 13.06-fold (95% CI, 8.08 to 21.11) increased risk for breakthrough infection with the Delta variant compared to those previously infected, when the first event (infection or vaccination) occurred during January and February of 2021” (Gazit et al., 2021). Ultimately, Gazit et al. (2021) demonstrate that “natural immunity confers *longer lasting and stronger protection against infection, symptomatic disease and hospitalization* caused by the Delta variant of SARS-CoV-2, compared to the BNT162b2 two-dose vaccine-induced immunity [my emphasis].”

Alfego et al. (2021) tracked SARS-CoV-2 antibody seropositivity across the United States using national data from patients tested by nucleic acid amplification (NAAT) and serologic assays. They found that in “specimens from 39,086 individuals with confirmed positive COVID-19 ... both S [i.e., spike glycoprotein] and N [i.e., nucleocapsid protein] SARS-CoV-2 antibody results offer an encouraging view of how long humans may have protective antibodies against COVID-19, with curve smoothing showing population seropositivity reaching 90% within three weeks, regardless of whether the assay detects N or S-antibodies” (Alfego et al., 2021). What these results suggest is that previously infected individuals develop long-lasting protective immunity against *multiple* viral proteins (i.e., both nucleocapsid and spike glycoproteins). As Hartley et al. (2021) note, “COVID-19 patients rapidly generate B cell memory to both the spike and nucleocapsid antigens following SARS-CoV-2 infection.” Conversely, SARS-CoV-2-naïve individuals vaccinated against SARS-CoV-2 develop antibodies against the spike glycoprotein *only*, which suggests a narrower band of immunity. This would explain why prior infection is more effective at preventing symptomatic infection/reinfection by the Delta variant than vaccination alone. See the figure below for a representation of the proteins against which natural infection confers immunity:



(Cascella et al. 2021)

In a study to determine the duration of antibody levels in individuals previously infected with SARS-CoV-2, Wei et al. (2021) used “representative data from 7,256 United Kingdom COVID-19 infection survey participants who had positive swab SARS-CoV-2 PCR tests from 26-April-2020 to 14-June-2021.” They estimated that “antibody levels associated with protection against reinfection likely last 1.5–2 years on

average, with levels associated with protection from severe infection present for several years” (Wei et al., 2021). These results compare favourably with neutralizing antibody levels post-vaccination in SARS-CoV-2-naïve individuals. For instance, Nordström et al. (2021) found that “Vaccine effectiveness of BNT162b2 against infection waned progressively from 92% (95% CI, 92-93, $P<0.001$) at day 15-30 to 47% (95% CI, 39-55, $P<0.001$) at day 121-180, and from day 211 and onwards no effectiveness could be detected (23%; 95% CI, -2-41, $P=0.07$).” Likewise, against severe infection, Nordström et al. (2021) found that “effectiveness [of ChAdOx1 nCoV-19, the Oxford/AstraZeneca vaccine] waned from 89% (95% CI, 82-93, $P<0.001$) at day 15-30 to 42% (95% CI, -35-75, $P=0.21$) from day 181 and onwards, with sensitivity analyses showing notable waning among men, older frail individuals, and individuals with comorbidities.” Similarly, in a study conducted in Israel, Goldberg et al. (2021) found that “the overall estimated level of protection from prior SARS-CoV-2 infection for documented infection is 94.8% (CI: [94.4, 95.1]); hospitalization 94.1% (CI: [91.9, 95.7]); and severe illness 96.4% (CI: [92.5, 98.3]).” Importantly, Goldberg et al. (2021) also note that their results call into question the need to vaccinate previously-infected individuals! There appears to be no justification for doing so if (1) naturally acquired immunological memory is widespread, and (2) it is at least as protective as vaccine-induced immunity, which are both true.

In the next section, I will discuss vaccine effectiveness and safety in more detail. For now, it is worth reiterating and explaining why natural immunity is generally superior to vaccine-induced immunity. Beyond antibody levels, which are a topic that proponents of mass vaccination and annual booster doses tend to fixate upon the most, there are other components of the immune system aside from circulating antibodies that indicate long-term protection against pathogens. As Cox & Brokstad (2020) note, “the absence of specific antibodies in the serum does not necessarily mean an absence of immune memory.” For instance, T and B lymphocytes continue to replicate post-infection and have been shown to be protective upon reinfection for upwards of 17 years (Le Bert et al., 2020). Indeed, even infection by other, non-SARS-CoV-2 coronaviruses has been shown to generate cross-reactive immunological memory against SARS-CoV-2. This is because, as Le Bert et al. (2020) note,

“SARS-CoV-2 belongs to the *Coronaviridae*, a family of large RNA viruses that infect many animal species. ... All of these coronaviruses trigger antibody and T cell responses in infected patients: however, antibody levels appear to wane faster than T cells. SARS-CoV-specific antibodies dropped below the limit of detection within 2 to 3 years [sic], *whereas SARS-CoV-specific memory T cells have been detected even 11 years after SARS* [sic]. As the sequences of selected structural and non-structural proteins are highly conserved among different coronaviruses (for example, NSP7 and NSP13 are 100% and 99% identical, respectively, between SARS-CoV-2, SARS-CoV and the bat-associated bat-SL-CoVZXC21 [sic]), we investigated whether cross-reactive SARS-CoV-2-specific T cells are present in individuals who resolved SARS-CoV, and compared the responses with those present in individuals who recovered from SARS-CoV-2 infection. We also studied these T cells in individuals with no history of SARS or COVID-19 or of contact with patients with SARS-CoV-2. Collectively these individuals are hereafter referred to as individuals who were not exposed to SARS-CoV and SARS-CoV-2 (unexposed donors) [my emphasis].”

Further, Le Bert et al. (2020) observed that “patients ($n = 23$) who recovered from SARS (the disease associated with SARS-CoV infection) possess long-lasting memory T cells that are reactive to the N protein of SARS-CoV 17 years after the outbreak of SARS in 2003; these T cells displayed robust cross-

reactivity to the N protein of SARS-CoV-2.” In other words, those who recovered from SARS more than 17 years ago, even though they were unexposed to SARS-CoV-2, demonstrated robust cross-reactive T Cell-mediated immune responses to SARS-CoV-2, at least partially due to the genetic and structural similarities between SARS-CoV and SARS-CoV-2. SARS-CoV and SARS-CoV-2 share 79–80% genome sequence identity (Wang et al., 2020). Hence, the test subjects’ immune systems ‘recognized’ SARS-CoV-2 as sufficiently similar to SARS-CoV and then deployed memory T cells to neutralize it before it could replicate and cause disease. Crucially, in completely unexposed donors, Le Bert et al. (2020) “detected SARS-CoV-2-specific T cells in individuals with no history of SARS, COVID-19 or contact with individuals who had SARS and/or COVID-19 ($n = 37$).” What these findings suggest is that “infection with betacoronaviruses induces multi-specific and long-lasting T cell immunity against the structural N protein” (Le Bert et al., 2020).

With respect to protection against severe disease and infection by betacoronaviruses (including SARS-CoV-2), these findings run contrary to the view that natural protection is somehow non-robust or extremely short-lived and that annual or semi-annual booster vaccinations are necessary to keep up protection. Naturally acquired immunity is now very, very widespread, given the sheer number of mild or asymptomatic SARS-CoV-2 infections that have occurred and the overall non-severity of the COVID-19 for the general population. For instance, in another study, Le Bert et al. (2021) subsequently found that “asymptomatic SARS-CoV-2-infected individuals are not characterized by weak antiviral immunity; on the contrary, they mount a highly functional virus-specific cellular immune response.” Hence, one does not need to suffer a debilitating infection by SARS-CoV-2 to retain immunity long-term. Protection against SARS-CoV-2 can also result from infection by other betacoronaviruses that induce cross-reactive immunity, where ‘cross-reactive’ just refers to the immune system’s ability to recognize and neutralize new pathogens due to exposure to other, genetically similar pathogens (Frank, 2002). Evidence for cross-reactive protection is widespread. Mateus et al. (2020) note that “SARS-CoV-2-reactive CD4⁺ T cells have been reported in unexposed individuals, suggesting pre-existing cross-reactive T cell memory in 20 to 50% of people [my emphasis].” Furthermore, Ansari et al. (2021) “identified SARS-CoV-2 cross-reactive CD4⁺ T cells in around 66% of the unexposed individuals [$n = 42$].” Consider the following:

“In this study, we have examined the traits and stability of immune memory in unexposed donors and patients recovered from mild COVID-19. We show that the SARS-CoV-2 cross-reactive antibodies and CD4⁺ T cells exist in the unexposed donors, with Non-spike domains as the predominant target of CD4⁺ T cells in ~66% of the individuals. Moreover, we also show that immunological memory to SARS-CoV-2 is detectable in mild COVID-19 patients up to 5 months (median ~3 months) after recovery both in the CD4⁺ T cells and B cells. Interestingly, the durable immune memory in COVID-19 patients was highly targeted towards the Spike glycoprotein of the SARS-CoV-2. Our work provides the evidence of pre-existing reactivity and immune memory detectable in mild COVID-19 patients from the geographical location that is experiencing high burden of SARS-CoV-2 pandemic with an extremely low case fatality.” (Ansari et al., 2021).

Hence, given that there have occurred billions of SARS-CoV-2 infections worldwide, and given that mild to asymptomatic SARS-CoV-2 infection is sufficient to generate protective immunological memory for many years, it follows that most people (vaccinated or not) have protective immunity against SARS-CoV-2 (Ioannidis, 2021; Le Bert et al., 2020; Ansari et al., 2021). The fact that protective immunity is widespread explains the extremely low rates of reinfection and relatively low IFR for the vast majority of people (Abu-Raddad et al., 2021; Axfors and Ioannidis, 2021).

There is a plethora of evidence to indicate the existence of immunological memory from natural infection. From a meta-analysis examining data from fifty-four studies spanning 18 countries with a total of 12,011,447 individuals, followed up to 8 months after recovery from SARS-CoV-2 infection, Chivese et al. (2021) found that at 6 to 8 months post-infection, “the prevalence of detectable SARS-CoV-2 specific immunological memory remained high; IgG – 90.4% ... pooled prevalence of reinfection was 0.2% (95%CI 0.0 – 0.7, $I^2 = 98.8$, 9 studies).” Overall, Chivese et al. (2021) found that “Individuals who recovered from COVID-19 had an 81% reduction in odds of a reinfection (OR 0.19, 95% CI 0.1 – 0.3, $I^2 = 90.5\%$, 5 studies).” Gallais et al. (2021) studied individuals in households who recovered from mild COVID-19. All recovered individuals “developed anti-SARS-CoV-2 antibodies and a significant T cell response detectable up to 69 days after symptom onset” (Gallais et al., 2021). Gallais et al. (2021) also conclude that “T cell responses may be more sensitive indicators of SARS-CoV-2 exposure than antibodies ... [and so] *epidemiological data relying only on the detection of SARS-CoV-2 antibodies may lead to a substantial underestimation of prior exposure to the virus* [my emphasis].” Indeed, to reiterate a previous point, many attempt to justify annual booster vaccinations by pointing to the fact that antibodies tend to dissipate after a few months (Devlin and Kollwe, 2021). It would be incorrect to equate dissipating antibodies with a total lack of protection, and it would be incorrect to conclude from antibody detection alone what proportion of certain populations have been previously infected. Testing for T Cell and B Cell reactivity as well as serological tests are necessary for painting a more accurate picture of the overall level of protection in the population.

In a study that examined uninfected residents of Vancouver, British Columbia, Majdoubi et al. (2021) used a “highly sensitive multiplex assay and positive/negative thresholds established in infants in whom maternal antibodies have waned ... [and found that] *more than 90% of uninfected adults showed antibody reactivity against the spike protein, receptor-binding domain (RBD), N-terminal domain (NTD), or the nucleocapsid (N) protein from SARS-CoV-2* [my emphasis].” The prevalence of naturally acquired and/or pre-existing immunity likely involves the fact that “T cells recognizing peptides conserved among coronaviruses are more abundant and tend to have a “memory” phenotype compared with those unique to SARS-CoV-2” (Mallajosyula et al., 2021). Again, what this means is that infection confers immunity to multiple different proteins (e.g., spike, nucleocapsid, etc.) whose structures are conserved across multiple distinct betacoronaviruses. T cells that recognize these proteins, as Mallajosyula et al. (2021) note, are more abundant and tend to last longer than those that are highly specific to SARS-CoV-2, e.g., those induced by vaccination in SARS-CoV-2-naïve individuals. These findings are confirmed by Moderbacher et al. (2021) who conclude that “Coordinated SARS-CoV-2-specific adaptive immune responses were associated with milder disease, suggesting roles for both CD4⁺ and CD8⁺ T cells in protective immunity in COVID-19.” This could further explain why naturally acquired immunity tends to perform at least as well and, according to some studies, even better than vaccine-induced immunity in SARS-CoV-2-naïve individuals. Likewise, “CD8⁺ T cells with these conserved specificities are *much more abundant in patients with mild COVID-19* [i.e., the majority of cases] versus those with a more severe illness, suggesting a protective role [my emphasis]” (Mallajosyula et al., 2021).

To sum up this section, naturally acquired immunity is widespread and *at least* as protective as vaccine-induced immunity. Mass vaccination is, therefore, unjustified. Now, in light of all this, I should not need to persevere on the negative aspects of vaccination. I will do so, however, given that a proper understanding of adverse events (AEs), vaccine effectiveness, and research integrity is necessary for capturing the complete risk-benefit profile of vaccination against SARS-CoV-2.

Premise 3: The currently available SARS-CoV-2 vaccines are insufficiently tested, relatively poor quality, and unsafe for at least some demographics

Evidently, people should not generally receive pharmaceutical products that are insufficiently safety tested. Still, in response to a ‘crisis’, one might argue that people should receive insufficiently safety tested pharmaceutical products if they are poised to confer some benefit and if they do not immediately indicate unacceptable levels of risk (i.e., levels of risk that exceed those associated with the ‘crisis’). Then again, as I have already demonstrated, COVID-19 is not a crisis, and so it does not demand the mass deployment of insufficiently safety tested pharmaceutical products to literally every person on Earth, regardless of age and morbidity status. Of course, this view runs contrary to the views of some high-profile vaccine stakeholders. Bill Gates, for instance, thinks that “the only way to end the pandemic for good is to offer a vaccine to almost all of the planet’s 7 billion inhabitants” (Boyle, 2020). Given the evidence surveyed thus far, this must be one of the least evidence-based statements ever made. Likewise, pharmaceutical products obviously should not be mass deployed if they are of relatively poor quality with respect to the goals they are supposed to accomplish, e.g., “zero COVID” (Normile, 2021). Lastly, if there are risk signals that portend especially harmful long-term effects (e.g., cardiac complications) for certain demographics who are generally not at risk from the virus (e.g., younger and/or immunocompetent people), then the pharmaceutical products in question should not be mass deployed. All three of the foregoing considerations apply to the current SARS-CoV-2 vaccines. They are (1) insufficiently safety tested, (2) relatively poor quality, and (3) risky for demographics who are not at risk from COVID-19. Therefore, they should not be mass deployed, and should probably be discontinued indefinitely.

1. Insufficient Testing

When I say, ‘insufficiently tested’, I mean two things: (1) research has not gone on long enough to determine the vaccines’ effectiveness/safety over time and for certain demographics, and (2) the research is of extremely poor quality, particularly the pharmaceutical industry-sponsored clinical trials. We should not view the currently available clinical evidence for the effectiveness/safety of the SARS-CoV-2 vaccines as much more than marketing. To illustrate, consider this recent piece from the *British Medical Journal (BMJ)*:

“But, for researchers who were testing Pfizer’s vaccine at several sites in Texas during that autumn, speed may have come at the cost of data integrity and patient safety. A regional director who was employed at the research organisation Ventavia Research Group has told *The BMJ* that the *company falsified data, unblinded patients, employed inadequately trained vaccinators, and was slow to follow up on adverse events reported in Pfizer’s pivotal phase III trial*. ... After repeatedly notifying Ventavia of these problems, the regional director, Brook Jackson, emailed a complaint to the US Food and Drug Administration (FDA). *Ventavia fired her later the same day*. Jackson has provided *The BMJ* with dozens of internal company documents, photos, audio recordings, and emails. ... Ventavia was not keeping up with data entry queries, shows an email sent by ICON, the contract research organisation with which Pfizer partnered on the trial. ... [Problems included, but were not limited to] ...

- Participants placed in a hallway after injection and not being monitored by clinical staff
- *Lack of timely follow-up of patients who experienced adverse events*

- Protocol deviations not being reported
- Vaccines not being stored at proper temperatures
- Mislabelled laboratory specimens, and
- Targeting of Ventavia staff for reporting these types of problems [my emphasis].” (Thacker, 2021).

Across multiple clinical trial sites managed by Ventavia, these problems were systemic. Among the documents leaked to the *BMJ* by the Ventavia whistleblower were documents listing various ‘action items’ that required immediate attention (Demasi, 2021). One of the action items pertaining to AE reports is particularly troubling. Note the highlighted portion in the screenshot below:

From: [REDACTED]
 To: [REDACTED]
 Subject: Common findings/action items all sites
 Date: Monday, September 21, 2020 9:33:27 PM
 Attachments: [image001.png](#)
[image002.png](#)

This is really rough- I had so many distractions trying to type this up, but hopefully, it's a helpful start:

Finding of Concern:	Details of Findings:	Solution (QUICK FIX):	Comments:
HIPAA Violations	Sites not consistent with protecting PHI- PHI left on desks (FW front desk example); patient schedule with patient names left on counter faced up; patient folders out on counters in the clinic and faced up with names visible; emails with patient names in the subject line.	1. RDs to observe and correct at sites when onsite- discuss/re-educated with employees at the site as concerns are found. 2. "Audit Readiness" training- I think we still need this for sites quickly, and we HIPAA can be included.	Where are we with HIPPS training?
Outdated Protocol Pages in Patient Folders	Sites are not keeping the Schedule of Events pages in the patient folders current and some copies are so poorly printed that they are completely illegible, defeating the purpose of them being placed in the chart.	1. Pull all protocol pages out of all charts. 2. Reassess the process for future studies.	
AEs Not Reported	AEs from diary entries are not being reported correctly or at all- 1. Protocol is unclear; 2. Conflicting information received from Pfizer	1. We need to get an official direction from Pfizer. Email from ICON received but the email was not clear as well and not an official "Clarification" or "Addendum" or even a "Memo".	Email from ICON dated 8/17/20. FUTURE: revise AE log

If AEs were ‘not being reported correctly or at all’, and if this problem was not unique to this particular trial site (and we have no reason to think that it would be), then none of the clinical trial results paint an accurate picture of the risks and benefits associated with receiving the Pfizer-BioNTech vaccine. Without an accurate picture of the risks and benefits, individuals cannot make sound decisions and, therefore, cannot give their full informed consent to receive the Pfizer-BioNTech vaccine. We likewise have little reason to think these problems with the quality of the clinical research were unique to the Pfizer-BioNTech trials. Consider the fact that Ventavia is just one of many contract research organizations (CROs) that the current vaccine manufacturers – Pfizer-BioNTech, Oxford/AstraZeneca, Johnson & Johnson, etc. – hired to conduct their trials. CROs are notorious for designing and conducting clinical trials in ways that principally benefit their clients (i.e., the pharmaceutical industry) at the cost of scientific integrity (McHenry & Jureidini, 2020). Consider the following:

“The companies hire contract research organizations (CROs) to conduct the clinical trials, academic researchers to design the trials and act as clinical investigators, medical communication companies to ghostwrite the publications, and public relations firms to create positive images of the drugs for prescribing physicians and the public. All are eager to serve their client’s best interest, irrespective of whether the drugs being evaluated are the latest miracles of science. Almost always, they are not.

...

Because the companies invest enormous sums to bring new drugs onto the market, failure is not an option. Aside from the suppression of negative data in publication results, the very design of the trial is often manipulated in subtle ways that escape detection in the peer-review process. Common strategies for ensuring success include conducting the trial drug against a treatment known to be inferior, testing it against too low (or too high) a dose of the competitor drug, and excluding placebo responders in the pretreatment, wash-out phase of the trial.” (McHenry & Jureidini, 2020).

Bias at this level is possible only because industry controls that vast majority of clinical drug trials. To illustrate, consider that “at least three of the world’s largest advertising agencies own not only medical communication companies, but also contract research organizations (CROs)” (Sismondo and Doucet, 2010). Hence, the advertisers who advertise drugs for pharmaceutical companies *own the firms that conduct the research to determine the safety/efficacy of those drugs!* Likewise, up to 75% of clinical drug trials are funded in whole or in part by pharmaceutical companies (Yaphe et al., 2001). Industry funding has also been associated with a greater percentage of ‘positive’ outcomes that weigh in favour of the safety/efficacy of pharmaceutical products (Yaphe et al., 2001). For instance, Yaphe et al. (2001) observed the following proportions of industry-funded randomized controlled trials (RCTs) published in the ‘Big 5’ medical journals between 1992–1994:

TABLE 1 Distribution of randomized controlled drug studies by journal, 1992–1994 (n = 314)

Journal	No. of studies	Studies with industry funding
<i>New England Journal of Medicine</i>	116	70 (60%)
<i>Lancet</i>	90	61 (67%)
<i>Annals of Internal Medicine</i>	54	38 (70%)
<i>British Medical Journal</i>	28	21 (75%)
<i>Journal of the American Medical Association</i>	26	19 (73%)

Yaphe et al. (2001) also observed the following association between industry funding and positive results:

TABLE 2 Outcome of randomized controlled drug trials in five general interest medical journals 1992–1994 by source of funding (n = 305)

	No. of studies (with raw percentages)	
	Positive outcome	Negative outcome
Industry funding	181 (87%)	28 (13%)
Non-industry funding	62 (65%)	34 (35%)

This table excludes the nine studies from Table 1 with ‘uncertain’ outcome.

As a result of near-complete industry control of the science pertaining to pharmaceutical products, examples of corruption in pharmaceutical industry-sponsored clinical research are abound. Indeed, given that pharmaceutical companies *own* the raw data for their trials, they typically do not make the data available to independent third-parties unless they are legally pressured to do so (Spinney, 2020). As such, many studies make it through the peer-review processes of mainstream medical journals, despite using fraudulent data or hopelessly biased research methods that ultimately favour industry at the patients’ expense.

A particularly notorious example of corruption in industry-sponsored research would be GlaxoSmithKline’s (GSK’s) infamous “Study 329” (McHenry & Jureidini, 2020). In 2001, the pharmaceutical company GSK (formerly SmithKline Beecham) funded an RCT widely known as Study 329 to determine the safety/efficacy of paroxetine, one of their selective serotine reuptake inhibitors (SSRIs) (McHenry & Jureidini, 2020). GSK intended to secure regulatory approval from the FDA to market paroxetine as a drug for treating adolescent depression. Study 329 was designed with the intent to conceal serious AEs. When there was a clear risk signal of increased suicidality in the treatment arm, the researchers chose to code suicidality euphemistically as “emotional lability” (McHenry & Jureidini, 2020). To illustrate the effect that this could have on peer reviewers, imagine if in a trial for a particular drug, there was a marked increase in myocardial infarction in the treatment arm compared to the placebo arm. If researchers were to code myocardial infarction as ‘chest discomfort’, or something else that sounds relatively innocuous, then the risk-benefit ratio of the drug would be compromised in that it would make the drug appear safer than it really is. Evidently, myocardial infarction ≠ chest discomfort. Coding suicidality as ‘emotional lability’ has the same effect.

Ultimately, more than a decade later, and upon reanalysis by independent researchers, paroxetine was found to *increase* suicidal ideation in adolescents (Spinney, 2020). Also, upon reanalysis, the effects of paroxetine on adolescent depression were found to be “not statistically different from placebo for any prespecified primary or secondary outcome measure” (Doshi, 2015). To date, Study 329 has not been retracted. Peter Doshi (2015), editor of the *BMJ*, sums up the situation succinctly:

“It is often said that science self corrects. But for those who have been calling for a retraction of the Keller paper for many years, the system has failed. None of the paper’s 22 mostly academic university authors, nor the journal’s editors, nor the academic and professional institutions they belong to, have intervened to correct the record. The paper remains without so much as an erratum, and none of its authors – many of whom are educators and prominent members of their respective professional societies – have been disciplined.” (Doshi, 2015).

The intransigence of the authors, the journal, the universities, regulators, and the drug companies stems from the fact that they routinely work in concert to promote drugs that are either ineffective, unsafe, or both. Due to their mutual ties and support for each other, they rarely face punitive consequences. Medical journals are incentivized to publish and *never retract* studies like Study 329, even upon independent reanalysis, because they stand to make millions from reprints that they sell to medical professionals (McHenry & Jureidini, 2020). As McHenry & Jureidini (2020) note, “A single article reporting on a blockbuster drug (defined by industry as a drug with annual sales of more than US\$1billion) can net the journal over a million dollars in reprint sales.” Universities also benefit in that they profit from the clinical trial revenue, and their researchers have little incentive to call for retractions given that increasing their publication numbers adds to their credentials (McHenry & Jureidini, 2020). Regulators are compromised in that their review boards are often composed of former reps for the pharmaceutical industry. The FDA has often been criticized for its ‘revolving door’ model of hiring (Piller, 2018). Consider the following from Piller (2018):

“FDA staffers play a pivotal role in drug approvals, presenting evidence to the agency's advisory panels and influencing or making approval decisions. They are free to move to jobs in pharma, and many do; in a 2016 study in *The BMJ*, researchers examined the job histories of 55 FDA staff who had conducted drug reviews over a 9-year period in the hematologyoncology field. They found that 15 of the 26 employees who left the agency later worked or consulted for the biopharmaceutical industry.”

Former FDA officials who go to work for the pharmaceutical industry often maintain close connections with FDA staffers. As such, corruption is pervasive. Indeed, salient examples of the revolving door model are pervasive in the COVID-era. For instance, Scott Gottlieb was the commissioner of the FDA from 2017–2019 (AEI, 2021). Before that, he was a member of the product investment board at GSK from 2010–2017, the same company that funded Study 329 (LinkedIn, 2021). Today, Gottlieb is on the board of directors at Pfizer (AEI, 2021). He frequently appears on the nightly news to market Pfizer’s COVID-19 vaccine and other drugs where he is routinely referred to by hosts as the ‘former FDA commissioner’ and, notably, *not* as a member of Pfizer’s board of directors (CNBC Television, 2021). Most recently, he has been marketing Pfizer’s booster vaccinations as highly protective against the Omicron variant, albeit without any reliable data to suggest as much (CNBC Television, 2021).

Unfortunately, all of the clinical trials conducted to determine the safety/efficacy of the currently available SARS-CoV-2 vaccines fall prey to all of the biases and industry corruption illustrated above. The issues extend well beyond those at the Ventavia trial site and into the very design of the clinical trials. As noted by Gøtzsche (2021), “Pfizer’s trial protocol contains a remarkable statement: During the 7 days following each vaccination, potential COVID-19 symptoms that overlap with specific systemic events (i.e., fever, chills, new or increased muscle pain, diarrhea, vomiting) should not trigger potential COVID-19 illness visit unless, in the investigator’s opinion, the clinical picture is more indicative of possible COVID-19 illness than vaccine reactogenicity.” Further, the protocol continues, “If, in the investigator’s opinion the symptoms are considered more likely to be vaccine reactogenicity, but a participant is required to demonstrate that they are SARS-CoV-2-negative, a local SARS-CoV-2 test may be performed.” (Gøtzsche, 2021). As Gøtzsche (2021) notes, given that the vaccines and COVID-19 can induce similar symptoms, these protocols could “mask symptoms of COVID-19” and thereby inflate the true efficacy of the Pfizer-BioNTech vaccine for preventing symptomatic disease. Again, as we have seen, clinical trial investigators are incentivised to opine that their benefactors’ products are efficacious,

and so the contract researchers' latitude in distinguishing vaccine reactogenicity from symptomatic COVID-19 could introduce significant biases.

Also, with respect to bias due to industry corruption, consider this:

"Serious adverse events were extremely poorly reported [during the Pfizer-BioNTech trials]. Apart from deaths, the trial report mentioned only those considered related to the vaccine ...

The most important number are ALL patients with serious adverse events, not just a tiny fraction of them (2%) that investigators, most of whom were on Pfizer's payroll, opined were related to the drugs. A supplement showed that 18 of the 29 authors of the trial report had received personal fees from Pfizer and 15 held stock in Pfizer. Only 8 authors (28%) had not declared any conflicts of interest. We do not expect authors who are so conflicted to report honestly on the harms they found, which they didn't do, either." (Gøtzsche, 2021).

To reiterate, reporting AEs in ways that clearly mask their prevalence or discount their severity paints a distorted picture of a pharmaceutical product's risk-benefit profile, thereby making it impossible for recipients to give their full informed consent to treatment. The medical journal article for Pfizer's clinical trials that has been most widely circulated to the public and physicians was obscure in its reporting of AEs, but a supplement to the article showed that "240 patients (1.1%) had severe adverse events on the vaccine versus 139 (0.6%) on placebo" (Gøtzsche, 2021). According to Gøtzsche (2021), "Pfizer did not provide a p-value, but [by Gøtzsche's calculations] $p = 2 \times 10^{-7}$... *The number needed to vaccinate (NNTV) in order to harm one patient severely was therefore 200.*" I will return to the concept of NNTV shortly when I discuss vaccine effectiveness in more detail. For now, just know that for every 200 individuals vaccinated with the Pfizer-BioNTech vaccine, one individual (who would have likely been fine otherwise) will incur a severe AE where 'severe' just means 'prevents daily activity and/or requires medical attention, hospitalization, etc.' (Gøtzsche, 2021).

Another way that pharmaceutical industry-sponsored researchers conceal AEs in order to boost the perceived safety/efficacy of their products is by testing them against a high dose of an active comparator (i.e., not a placebo) that *will itself have harmful AEs* (McHenry & Jureidini, 2020). The Oxford/AstraZeneca trials employed this method. In one of their clinical trials, "3,755 participants received the vaccine and 3,804 a meningococcal vaccine" (Gøtzsche, 2021). A meningococcal vaccine, depending especially on the dose administered, will also lead to AEs in some number of recipients. Therefore, selective reporting of AEs combined with testing against an active comparator that is more likely to lead to AEs in the control group will produce results that favour the treatment. Likewise, for the Oxford/AstraZeneca trials, "There are no data on numbers of patients with severe adverse events (those preventing daily activity), only on the numbers of such events, 84 [in the vaccine group] vs. 91 [in the control group]." Therefore, the contract researchers concluded, "the vaccine did not increase adverse events, but the trials were not adequate to study vaccine harms, *as 86% of the patients in the control groups received another vaccine and not a placebo* [my emphasis]" (Gøtzsche, 2021).

Similarly, for the NIH-Moderna vaccine, the frequency of AEs was presented misleadingly. The article coded severe AEs as "Grade 3 [AEs] ... defined on page 160 as preventing daily activity and requiring intensive therapeutic treatment" (Gøtzsche, 2021). The ratio of grade 3 events in the vaccine group [$n =$

14,677] vs. the control group [n = 14, 566] was 2,325 (15.8%) to 282 (1.9%) (Gøtzsche, 2021). Grade 4 events required emergency room visits, and the ratio for grade 4 events was 14 (0.1%) to 3 (0.1%) for the vaccine group and the control group, respectively (Gøtzsche, 2021). Hence, from these ratios alone we can conclude that for roughly every 7 patients vaccinated with the NIH-Moderna vaccine, “*one is severely harmed and unable to perform daily activities* [my emphasis]” (Gøtzsche, 2021). Evidently, this is not something that most people considered when determining whether they would receive the NIH-Moderna vaccine. The likelihood for most people that SARS-CoV-2 will leave them severely harmed, hospitalized, or unable to perform daily activities is obviously less than 1 in 7.

Beyond protocols that are likely designed to conceal AEs and boost perceived effectiveness, the trials are also limited by design (that is, by their endpoints) in terms of what they can tell us about the long-term effectiveness of the vaccines. Consider the following from Doshi (2020):

“Peter Hotez, dean of the National School of Tropical Medicine at Baylor College of Medicine in Houston, said, “Ideally, you want an antiviral vaccine to do two things . . . first, reduce the likelihood you will get severely ill and go to the hospital, and two, prevent infection and therefore interrupt disease transmission.”

Yet the current phase III trials are not actually set up to prove either [sic]. None of the trials currently under way are designed to detect a reduction in any serious outcome such as hospital admissions, use of intensive care, or deaths. Nor are the vaccines being studied to determine whether they can interrupt transmission of the virus.”

Indeed, the primary endpoint of most of the trials was whether participants (1) tested positive for SARS-CoV-2 via RT-PCR test, and/or (2) developed one of a number of COVID-19 symptoms (e.g., headache, fever, chills, cough, etc.) (Doshi, 2020). Researchers only followed up with trial participants for six months at the most, and nearly all of the unvaccinated controls have now been vaccinated outside of the trials. Hence, there will be no long-term follow-up or comparisons between the vaccinated and unvaccinated trial participants. It is worrying, therefore, that Walensky et al. (2021) could so brazenly lie in a published medical journal article by claiming that “Clinical trials have shown that the vaccines authorized for use in the US are highly effective against COVID-19 infection, severe illness, and death.” Six month follow-up data from Pfizer’s clinical trials clearly demonstrate otherwise. Consider the following:

Reported Cause of Death ^a	BNT162b2 (N=21,924) n	Placebo (N=21,921) n
Deaths	15	14
Acute respiratory failure	0	1
Aortic rupture	0	1
Arteriosclerosis	2	0
Biliary cancer metastatic	0	1
COVID-19	0	2
COVID-19 pneumonia	1	0
Cardiac arrest	4	1
Cardiac failure congestive	1	0
Cardiorespiratory arrest	1	1
Chronic obstructive pulmonary disease	1	0
Death	0	1
Dementia	0	1
Emphysematous cholecystitis	1	0
Hemorrhagic stroke	0	1
Hypertensive heart disease	1	0
Lung cancer metastatic	1	0
Metastases to liver	0	1
Missing	0	1
Multiple organ dysfunction syndrome	0	2
Myocardial infarction	0	2
Overdose	0	1
Pneumonia	0	2
Sepsis	1	0
Septic shock	1	0
Shigella sepsis	1	0
Unevaluable event	1	0

Table S4 | Causes of Death from Dose 1 to Unblinding (Safety Population, ≥16 Years Old). a. Multiple causes of death could be reported for each participant. There were no deaths among 12–15-year-old participants.

(Thomas et al., 2021).

The table above is presented in the Supplementary Appendix of Thomas et al. (2021), that is, *where it is difficult to find*. Clearly, if one more trial participant died in the treatment group than in the control group (i.e., 15 vs. 14), then the Pfizer-BioNTech trials do not demonstrate that the Pfizer-BioNTech vaccine is highly effective against “severe illness, and death” (Walensky et al., 2021). Severe illness and death were not even relevant endpoints to begin with. Likewise, it is somewhat worrying that the incidence rate of cardiac arrest is *4x greater in the treatment group than in the control group*. I will return to this later on in my discussion of the known AEs associated with the currently available SARS-CoV-2 vaccines. Severe cardiac events appear to be more common than initially anticipated.

The trials also suffered from outcome reporting bias. The FDA advises that clinical researchers present information pertaining to risk reduction from treatments as follows: “Provide absolute risks, not just relative risks. *Patients are unduly influenced when risk information is presented using a relative risk approach; this can result in suboptimal decisions.* Thus, an absolute risk format should be used [my emphasis].” (Brown, 2021). At least when it came to advertising the results of the vaccines’ clinical trials, vaccine effectiveness was presented to the public primarily in terms of relative risk reduction (RRR) (e.g., 95% effective). Subsequent analyses of the clinical trial data show that absolute risk reduction (ARR) paints a more accurate picture of the overall effectiveness of the vaccines for reducing disease at the population level. ARR should play a more significant part in decision-making surrounding mass vaccination campaigns as it conveys a more realistic picture of just how many people will *benefit* from receiving a SARS-CoV-2 vaccine. Consider the following from Brown (2021):

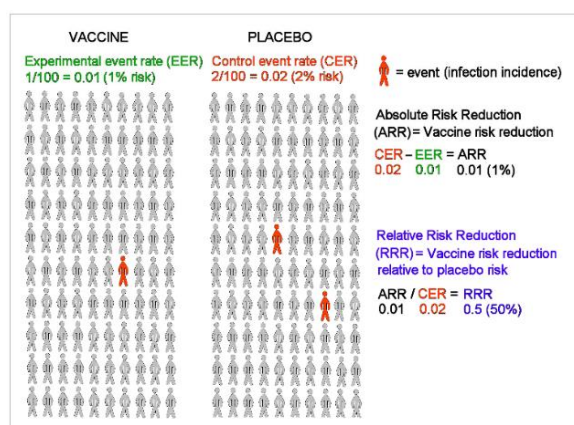
“Absolute risk reduction (ARR) and relative risk reduction (RRR) are measures of treatment efficacy reported in randomized clinical trials. Because the ARR and RRR can be dramatically different in the same trial, it is necessary to include both measures when reporting efficacy outcomes to avoid outcome reporting bias. In the present article, a critical appraisal of publicly available clinical trial data verifies that *absolute risk reduction percentages for Pfizer/BioNTech vaccine BNT162b2 [sic] and Moderna vaccine mRNA-1273 [sic] are, respectively, 0.7%; 95% CI, 0.59% to 0.83%; $p = 0.000$, and 1.1%; 95% CI, 0.97% to 1.32%; $p = 0.000$.*

...

Reporting relative measures may be sufficient to summarize evidence of a study for comparisons with other studies, but absolute measures are also necessary for applying study findings to specific clinical or public health circumstances [sic]. *Omitting absolute risk reduction findings in public health and clinical reports of vaccine efficacy is an example of outcome reporting bias, which ignores unfavorable outcomes and misleads the public’s impression and scientific understanding of a treatment’s efficacy and benefits [sic].* Furthermore, the ethical and legal obligation of informed consent requires that patients are educated about the risks and benefits of a healthcare procedure or intervention [sic] [my emphasis].”

ARR represents the total “disease risk difference between the placebo and vaccine groups, i.e., the CER [control event rate] minus the EER [experimental event rate] ($2\% - 1\% = 1\%$). So, if the experimental event rate of symptomatic, laboratory-confirmed COVID-19 in the Pfizer-BioNTech treatment group was 0.036, and if the control event rate in the placebo group was 0.75, then the ARR equals 0.7 (i.e., $0.75 [\text{CER}] - 0.036 [\text{EER}] = 0.714$). Hence, we should expect to see a cumulative reduction in symptomatic COVID-19 of only 0.7% in fully vaccinated populations. The ARR for the SARS-CoV-2 vaccines is so low because the incidence rate of severe disease is also quite low. Infection is widespread, but, the

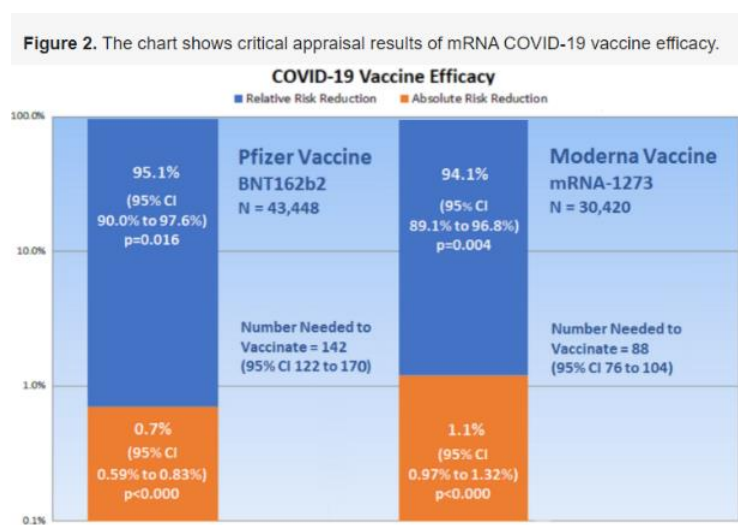
epidemiologically relevant outcomes that the vaccines are supposed to prevent are borne a very small percentage of the population. Hence, mass vaccination is poised to afford virtually zero benefit to those who, frankly, are not at risk. Therefore, ARR for the SARS-CoV-2 vaccines is extremely low. Consider the following representation of the cumulative disease risk difference between two hypothetical vaccinated and unvaccinated populations in terms of ARR:



(Brown, 2021).

Crucially, none of the vaccines' clinical trials reported on the number of persons needed to vaccinate in order to prevent one case of symptomatic COVID-19, sometimes abbreviated to NNV or NNTV. Once again, NNV is a relevant measurement for determining just how many people will benefit from administering a treatment on a mass scale (Hashim et al., 2015). It is "defined as the number of persons needed to vaccinate in order to prevent one outcome, and it combines both vaccine effectiveness and incidence of disease" (Hashim et al., 2015). If the NNV is particularly high for some demographics, then it might not be worth it for them to be vaccinated, especially if being vaccinated poses additional risks in terms of severe AEs. There are a number of estimates of the NNV for the currently available SARS-CoV-2 vaccines. Consider the following from Brown (2021):

"By comparison, **Figure 2** of the present article shows that the **NNV [number of persons needed to vaccinate to reduce one case]** for the Pfizer-BioNTech and Moderna vaccines are 142 (95% CI 122 to 170) and 88 (95% CI 76 to 104), respectively [my emphasis]." (Brown, 2021).



(Brown, 2021)

For the Pfizer-BioNTech vaccine, Brown (2021) calculates the NNV, for all age cohorts included in the trial, as 142 persons. For the Moderna vaccine, Brown (2021) calculates the NNV as 88. These numbers are calculated by dividing 1 (as in, one person) by the ARR, or, for the Pfizer-BioNTech and Moderna vaccines, respectively, $1/0.7$ and $1/1.1$. Others estimate higher, based on follow-up data regarding AAR from Dagan et al. (2021). Consider the following:

“The Number Needed To Vaccinate (NNTV) = 256 ($1/0.0039$), which means that to prevent just 1 Covid-19 case 256 individuals must get the vaccine; **the other 255 individuals derive no benefit, but are subject to vaccine adverse effects, whatever they may be and whenever we learn about them** ... [my emphasis]” (BMJ, 2021).

Olliario et al. (2021) also note the following:

“ARR is also used to derive an estimate of vaccine effectiveness, which is the number needed to vaccinate (NNV) to prevent one more case of COVID-19 as $1/ARR$. NNVs bring a different perspective: 81 for the Moderna–NIH, 78 for the AstraZeneca–Oxford, 108 for the Gamaleya, 84 for the J&J, and 119 for the Pfizer–BioNTech vaccines. *The explanation lies in the combination of vaccine efficacy and different background risks of COVID-19 across studies: 0.9% for the Pfizer–BioNTech, 1% for the Gamaleya, 1.4% for the Moderna–NIH, 1.8% for the J&J, and 1.9% for the AstraZeneca–Oxford vaccines.*

ARR (and NNV) are sensitive to background risk—*the higher the risk, the higher the effectiveness*—as exemplified by the analyses of the J&J's vaccine on centrally confirmed cases compared with all cases: both the numerator and denominator change, RRR does not change (66–67%), but the one-third increase in attack rates in the unvaccinated group (from 1.8% to 2.4%) translates in a one-fourth decrease in NNV (from 84 to 64).

...

Uncoordinated phase 3 trials do not satisfy public health requirements; platform trials designed to address public health relevant questions with a common protocol will allow decisions to be made, informed by common criteria and uniform assessment. These considerations on efficacy and effectiveness are based on studies measuring prevention of mild to moderate COVID-19 infection; they were not designed to conclude on prevention of hospitalisation, severe disease, or death, or on prevention of infection and transmission potential. Assessing the suitability of vaccines must consider all indicators, and involve safety, deployability, availability, and costs.” (Olliario et al., 2021).

Background risks may be the most important metric for weighing the benefits and risks associated with vaccinating whole populations, i.e., “the higher the risk, the higher the effectiveness” (Olliario et al., 2021). Currently, the most popularly lauded metrics of vaccine effectiveness in the media and public discourse (e.g., 95%) only take into account the benefits from vaccination to those who are already at risk (i.e., a very small proportion of most populations). Consider the following:

“RRR focusses on those who benefit from the vaccine and, although more stable across event rates (levels of risk for COVID-19), its interpretation requires knowing the actual background risk and its variability. *Absolute risk reduction (ARR) considers all individuals and translates conveniently into number needed to vaccinate within a population with a given risk*, but its

public health significance varies with the risk. For any given RRR, ARR is higher when event rates are higher – e.g., earlier into a vaccination programme, or in high-risk groups – and decreases as risks decline.” (Olliaro et al., 2021).

Clearly, the full clinical picture of the vaccines’ effectiveness has not been communicated to the public. Many who are evidently not at any appreciable risk from SARS-CoV-2 whatsoever are being made to receive vaccines from which they derive no benefit. Likewise, with the integrity of the research thrown into question by revelations of scientific fraud, nobody should be forced to take any of the available SARS-CoV-2 vaccines.

2. Poor quality

The overall performance of the vaccines (that is, compared to their anticipated and/or advertised performance) has been lackluster to say the least. Even the overblown RRR has reduced significantly over time. In a cohort study conducted in Sweden, Björk et al. (2021) found that “The estimated vaccine effectiveness in preventing infection ≥ 7 days after second dose was 86% (95% CI 72-94%) *but only 42% (95% CI 14-63%) ≥ 14 days after a single dose* [my emphasis].” Conversely, Björk et al. (2021) also observed that “*a prior positive test was associated with 91% (95% CI 85 to 94%) effectiveness against new infection among the unvaccinated* [my emphasis].” (Björk et al., 2021). Hence, natural infection appears more effective. Maeda et al. (2021) report that “BNT162b2 efficacy is likely to be diminished to under detection limit by 6-7 months post-1st shot on average.” They note that “If BNT162b2-elicited immune memory is lost soon, additional vaccine(s) or other protective means would be needed” (Maeda et al., 2021). This partially explains the push for multiple booster doses. Alarming, Brosh-Nissimov et al. (2021) observed the following among fully vaccinated COVID-19 patients in a hospital in Israel:

“A total of 152 patients were included, accounting for half of hospitalized fully vaccinated patients in Israel. Poor outcome was noted in 38 patients and mortality rate reached 22% (34/152). Notably, the cohort was characterized by a high rate of co-morbidities predisposing to severe COVID-19, including hypertension (108; 71%), diabetes (73; 48%), congestive heart failure (41; 27%), chronic kidney and lung diseases (37; 24% each), dementia (29; 19%) and cancer (36; 24%), and only six (4%) had no co-morbidities. Sixty (40%) of the patients were immunocompromised. Higher viral load was associated with a significant risk for poor outcome. Risk also appeared higher in patients receiving anti-CD20 treatment and in patients with low titres of anti-Spike IgG, but these differences did not reach statistical significance.

...

We found that severe COVID-19 infection, associated with a high mortality rate, might develop in a minority of fully vaccinated individuals with multiple co-morbidities. Our patients had a higher rate of co-morbidities and immunosuppression compared with previously reported non-vaccinated hospitalized individuals with COVID-19. Further characterization of this vulnerable population may help to develop guidance to augment their protection, either by continued social distancing, or by additional active or passive vaccinations [my emphasis].” (Brosh-Nissimov et al., 2021).

Crucially, the vaccines were supposed to protect those most vulnerable from symptomatic infection. These data suggest that they may not. Unsurprisingly, elderly and severely immunocompromised people

were largely excluded from the clinical trials. Only 4.4% of the trial participants were aged 75+ years, which means that the only group of people that really stood to benefit from the vaccines is *highly underrepresented* in the trials (Kostoff et al., 2021). See the following:

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.

(Kostoff et al., 2021)

Hence, I would go so far as arguing that neither the clinical trials nor the post-marketing surveillance data show any solid evidence for the claim that the vaccines effectively protect against severe disease leading to hospitalization and death in the very elderly/immunocompromised. As Kostoff et al. (2021) note, “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* [my emphasis].”

Worse yet, there is evidence to suggest that the issues with vaccine performance could involve the degradation of the mRNA due to improper handling and storage at temperatures in excess of what is necessary for maintaining the integrity of the mRNA. Consider this from Tinari (2021):

“As it conducted its analysis of the Pfizer-BioNTech covid-19 vaccine in December, the European Medicines Agency (EMA) was the victim of a cyberattack. [sic] More than 40 megabytes of classified information from the agency’s review were published on the dark web, and several journalists – including from *The BMJ* – and academics worldwide were sent copies of the leaks. They came from anonymous email accounts and most efforts to interact with the senders were unsuccessful. None of the senders revealed their identity, and the EMA says it is pursuing a criminal investigation.

The BMJ has reviewed the documents, which show that *regulators had major concerns over unexpectedly low quantities of intact mRNA in batches of the vaccine developed for commercial production.*

EMA scientists tasked with ensuring manufacturing quality – the chemistry, manufacturing, and control aspects of Pfizer’s submission to the EMA – worried about “truncated and modified mRNA species present in the finished product.” Among the many files leaked to *The BMJ*, an email dated 23 November by a high ranking EMA official outlined a raft of issues. In short, commercial manufacturing was not producing vaccines to the specifications expected, and

regulators were unsure of the implications. EMA responded by filing two “major objections” with Pfizer, along with a host of other questions it wanted addressed.

...

RNA instability is one of the biggest hurdles for researchers developing nucleic acid based vaccines. It is the primary reason for the technology’s stringent cold chain requirements and has been addressed by encapsulating the mRNA in lipid nanoparticles (box).

“The complete, intact mRNA molecule is essential to its potency as a vaccine,” professor of biopharmaceutics Daan J.A. Crommelin and colleagues wrote in a review article in *The Journal of Pharmaceutical Sciences* late last year. “*Even a minor degradation reaction, anywhere along a mRNA strand, can severely slow or stop proper translation performance of that strand and thus result in the incomplete expression of the target antigen.*”

Crommelin and colleagues note that specific regulatory guidance for mRNA based vaccines has yet to be developed, and *The BMJ*’s attempts to clarify current standards were unsuccessful [my emphasis].” (Tinari, 2021).

In short, damaged mRNA in the Pfizer-BioNTech and Moderna vaccines could lead to the improper or incomplete production of the target antigens (i.e., synthetic spike glycoproteins) such that a properly protective immune response is not induced and immunity is not conferred. This could explain the decline in quality of mRNA-vaccine-induced immunity. It could also explain why, according to Saul et al. (2021), in their reanalysis of the data from the two dose regimen of the Pfizer-BioNTech vaccine, “at least in the short period covered by the timetable of the Phase III trial, *there was no additional benefit from a second vaccination.*” Coupled with improper handling, the two dose regimen could leave time for the mRNA to decay to the point where the second doses are useless, harmful, or both.

Perhaps most importantly, the currently available SARS-CoV-2 vaccines have no discernable effect on the transmission of SARS-CoV-2 or on SARS-CoV-2’s ability to infect and replicate within the upper respiratory tract. Hence, *both vaccinated and unvaccinated individuals are equally likely to be infectious.* Consider the following from Singanayagam et al. (2021):

“SAR [secondary attack rate] among household contacts exposed to fully vaccinated index cases was similar to household contacts exposed to unvaccinated index cases (25% [95% CI 15–35] for vaccinated vs 23% [15–31] for unvaccinated). 12 (39%) of 31 infections in fully vaccinated household contacts arose from fully vaccinated epidemiologically linked index cases, further confirmed by genomic and virological analysis in three index case-contact pairs. ... Vaccination reduces the risk of delta variant infection and accelerates viral clearance. Nonetheless, *fully vaccinated individuals with breakthrough infections have peak viral load similar to unvaccinated cases and can efficiently transmit infection in household settings, including to fully vaccinated contacts. Host–virus interactions early in infection may shape the entire viral trajectory* [my emphasis].”

The caveat in the above quote about vaccination reducing the risks of delta variant infection have been challenged. For instance, Riemersma et al. (2021) show that both vaccinated and unvaccinated individuals shed infectious virus at the same rate, suggesting that they are at least equally subject to

infection by the dominant strain of SARS-CoV-2, i.e., Delta, and are equally infectious. Consider the following:

“Ct values <25 were detected in 7 of 24 unvaccinated (29%; CI: 13-51%) and 9 of 11 fully vaccinated asymptomatic individuals [i.e., vaccinated asymptomatic people presented with higher viral loads] (82%; CI: 48-97%), and 158 of 232 unvaccinated (68%, CI: 62-74%) and 156 of 225 fully vaccinated (69%; CI: 63-75%) symptomatic individuals. *Testing a subset of these low-Ct samples revealed infectious SARS-CoV-2 in 15 of 17 specimens (88%) from unvaccinated individuals and 37 of 39 (95%) from vaccinated people.* Infectious virus was detected in the sole specimen tested from an asymptomatic fully vaccinated individual (**Supplemental Figure 2**). Although few asymptomatic individuals were sampled, these results indicate that even asymptomatic, fully vaccinated people might shed infectious SARS-CoV-2 [my emphasis].”

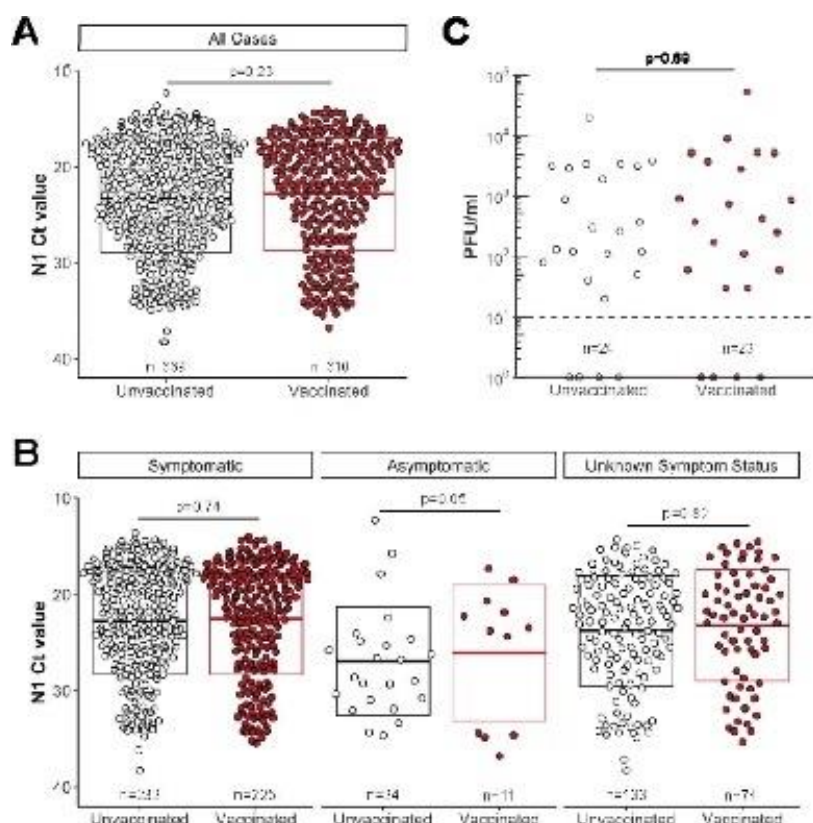


Figure 1. Individuals infected with SARS-CoV-2 despite full vaccination have low Ct values and shed similar amounts of infectious virus as uninfected individuals.

Recall that low Ct values correlate with high infectiousness (Stang et al. 2021; Singanayagam et al. 2020). Hence, vaccinated and unvaccinated individuals are equally infectious. Brown et al. (2021) observed patterns consistent with this hypothesis during an outbreak of SARS-CoV-2 traced back to a large public gathering where the majority of individuals in attendance were fully vaccinated, and in which fully vaccinated individuals were *overrepresented* among positive case counts. Consider this:

“During July 2021, 469 cases of COVID-19 associated with multiple summer events and large public gatherings in a town in Barnstable County, Massachusetts, were identified among Massachusetts residents; *vaccination coverage among eligible Massachusetts residents was*

69%. *Approximately three quarters (346; 74%) of cases occurred in fully vaccinated persons* (those who had completed a 2-dose course of mRNA vaccine [Pfizer-BioNTech or Moderna] or had received a single dose of Janssen [Johnson & Johnson] vaccine ≥ 14 days before exposure). Genomic sequencing of specimens from 133 patients identified the B.1.617.2 (Delta) variant of SARS-CoV-2, the virus that causes COVID-19, in 119 (89%) and the Delta AY.3 sublineage in one (1%). Overall, 274 (79%) vaccinated patients with breakthrough infection were symptomatic. *Among five COVID-19 patients who were hospitalized, four were fully vaccinated; no deaths were reported.* Real-time reverse transcription-polymerase chain reaction (RT-PCR) cycle threshold (Ct) values in specimens from 127 vaccinated persons with breakthrough cases were similar to those from 84 persons who were unvaccinated, not fully vaccinated, or whose vaccination status was unknown (median = 22.77 and 21.54, respectively) [my emphasis]. (Brown et al., 2021). ”

Crucially, if fully vaccinated individuals are overrepresented among positive case counts during outbreaks, then we have no reason to believe that they are somehow less infectious or substantially less subject to infection than unvaccinated individuals. Additionally, during a nosocomial (i.e., in hospital-located) outbreak of the Delta variant, Shitrit et al. (2021) observed the following:

“A nosocomial outbreak of SARS-CoV-2 Delta variant infected *42 patients, staff and family members; 39 were fully vaccinated.* The attack rate was 10.6% (16/151) among exposed staff and reached 23.7% (23/97) among exposed patients in a highly vaccinated population, 16-26 weeks after vaccination (median: 25 weeks). All cases were linked and traced to one patient. Several transmissions occurred between individuals wearing face masks. *Fourteen of 23 patients became severely sick or died, raising a question about possible waning immunity* [my emphasis].”

Again, this likely has something to do with the fact that the currently available SARS-CoV-2 vaccines do nothing to stop infection or viral replication in the upper respiratory tract, and so immune-compromised people (i.e., the only ones who theoretically stand to benefit the most from the vaccines) are not nearly as protected as many people might think. Consider that Acharya et al. (2021) “found no significant difference in cycle threshold values between vaccinated and unvaccinated, asymptomatic and symptomatic groups infected with SARS-CoV-2 Delta.” Perhaps the most incriminating of all bits of evidence is that fact that SARS-CoV-2 infection rates per 1,000,000 people by country *do not depend at all on vaccination rates* and, remarkably, slightly *increase* with higher rates of vaccination. Subramanian & Kumar (2021) recently observed the following results:

“At the country-level, there appears to be no discernable relationship between percentage of population fully vaccinated and new COVID-19 cases in the last 7 days (Fig. 1). In fact, the trend line suggests a marginally positive association such that countries with higher percentage of population fully vaccinated have higher COVID-19 cases per 1 million people. Notably, Israel with over 60% of their population fully vaccinated had the highest COVID-19 cases per 1 million people in the last 7 days. The lack of a meaningful association between percentage population fully vaccinated and new COVID-19 cases is further exemplified, for instance, by comparison of Iceland and Portugal. Both countries have over 75% of their population fully vaccinated and have more COVID-19 cases per 1 million people than countries such as Vietnam and South Africa that have around 10% of their population fully vaccinated.”

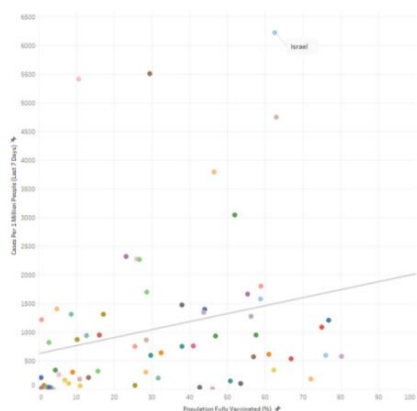


Fig 1 Relationship between cases per 1 million people (last 7 days) and percentage of population fully vaccinated across 68 countries as of September 3, 2021 (See Table S1 for the underlying data)

(Subramanian & Kumar, 2021)

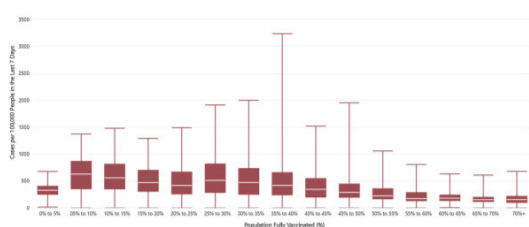


Fig 2 Median, interquartile range and variation in cases per 100,000 people in the last 7 days across percentage of population fully vaccinated as of September 2, 2021

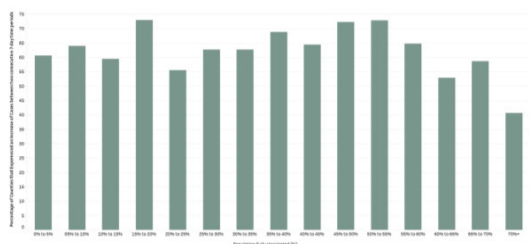


Fig 3 Percentage of countries that experienced an increase of cases between two consecutive 7-day time periods by percentage of population fully vaccinated across 2947 countries as of September 2, 2021

(Subramanian & Kumar, 2021)

These results are extremely troubling given that the vaccine rollouts were justified at least in part on the assumption that they would *reduce the overall spread of SARS-CoV-2*. They appear to be doing no such thing. Likewise, when observing these data, we must take into account the fact that observational case-positivity rates are being confounded by sampling bias. In other words, unvaccinated people are subject to routine testing more often than vaccinated people. This holds true from most social settings from schools, to workplaces, and even hospitals. Consider the following from Haas et al. (2021):

“Notably, Israel’s SARS-CoV-2 testing policy was different for unvaccinated and vaccinated individuals during the study period. At 7 days after the second dose, vaccinated individuals were exempt from the SARS-CoV-2 testing required of individuals who either had contact with a laboratory-confirmed case or returned from travel abroad. This testing policy might have resulted in a differential bias that would cause overestimation of vaccine effectiveness against asymptomatic infection (ie, asymptomatic people who received two doses were less likely to be tested than unvaccinated asymptomatic people). However, 19% of the 4.4 million PCR tests conducted during the study period were done on exempted individuals (MoH, unpublished data). Additionally, symptomatic individuals might have been reluctant to report symptoms for

fear of being blamed for infecting other individuals, in which case asymptomatic vaccine effectiveness would also be overestimated [my emphasis].” (Haas et al., 2021).

Note that, according to the Haas et al. (2021), only 19% of individuals tested were fully vaccinated during the study period, whereas the remaining 91% were not. Needless to say, if the vast majority of people subject to routine testing are unvaccinated, and if a positive test result within a certain period of time before hospitalization, death, etc., is sufficient (according to public policy) to diagnose COVID-19 as the probable cause of hospitalization or death, then official data will show more cases, deaths, hospitalizations, etc., among those who are not fully vaccinated *as a result of sampling bias*, and not due to greatly enhanced risk from being unvaccinated. Countries around the world are subjecting vaccinated and unvaccinated individuals to different routine testing protocols such that unvaccinated individuals will, in some cases, test positive more often than vaccinated individuals in certain settings, e.g., hospitals, long-term care (LTR) facilities. Consider the following policy set forth by the Ontario Ministry of Health (2021) regarding differential testing requirements for vaccinated and unvaccinated asymptomatic individuals in hospitals, LTR facilities, retirement homes, and so forth:

“Only asymptomatic individuals who are high-risk should be considered for molecular testing, including asymptomatic individuals who have received a positive antigen POCT or positive self-test kit result, and individuals from targeted testing groups ...

...

1.6 Guidance for Specific Settings

Facility Transfers

Examples of facility transfers include, but are not limited to:

- Admissions to hospital from another *hospital, long-term care home, retirement home or other congregate living setting/institution* (including group homes and equivalent higher-risk settings)
- Patients entering a residential treatment facility (e.g. a mental health or addiction program)
- Transfers from, or repatriation to community hospitals and regional tertiary/quaternary centres; or
- Transfers from an acute site to a post-acute site (e.g. patient transferred from hospital to complex continuing care/rehab) within a multi-site organization

...

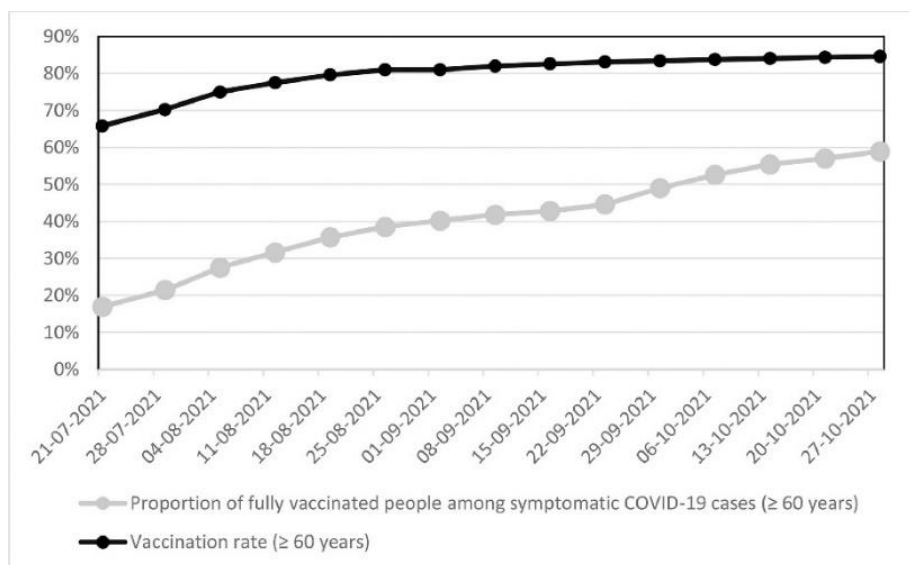
Testing of fully vaccinated individuals who are asymptomatic with no known COVID-19 exposure: As per PIDAC’s Interim Guidance on Infection Prevention and Control for Health Care Providers and Patients Vaccinated Against COVID-19 in Hospital and Long-Term Care Setting, ***testing is no longer recommended for asymptomatic fully vaccinated people who are being transferred. Individuals in the above settings who are not fully vaccinated can receive laboratory-based PCR testing for the purposes of facility transfers.*** Local PHUs and health organizations may make alternate testing recommendations/policies based on local epidemiology and within outbreak contexts. There is one exception to the above guidance: Newborn infants (<48 hours old at time of transfer) born to

individuals who are symptomatic and screen negative for symptoms: Such newborn infants should be considered exempt from routine COVID-19 testing on admission to destination facility.”

As one can clearly see from the above paragraph and from Haas et al. (2021), lopsided testing requirements for asymptomatic individuals will lead to disproportionate rates of test-positivity and ‘hospitalization’ among unvaccinated individuals, *regardless of vaccine effectiveness*. Anyone who is asymptomatic, unvaccinated, and in hospital and/or long-term care, a mental health facility, etc., *for any reason whatsoever* and who happens to test positive (despite being asymptomatic) will be counted among those hospitalized *because of* COVID-19, even when they are clearly not ill. Therefore, the observational data promulgated by the news media regarding differential rates of COVID-19 hospitalizations for vaccinated and unvaccinated populations is useless. From those data, we certainly cannot conclude that the vaccines are highly protective.

All this being said, even among *symptomatic* infections, the proportion of fully vaccinated individuals among them is rising exponentially every week. Consider the following from Kampf (2021):

“High COVID-19 vaccination rates were expected to reduce transmission of SARS-CoV-2 in populations by reducing the number of possible sources for transmission and thereby to reduce the burden of COVID-19 disease. Recent data, however, indicate that the epidemiological relevance of COVID-19 vaccinated individuals is increasing. In the UK it was described that secondary attack rates among household contacts exposed to fully vaccinated index cases was similar to household contacts exposed to unvaccinated index cases (25% for vaccinated vs 23% for unvaccinated). 12 of 31 infections in fully vaccinated household contacts (39%) arose from fully vaccinated epidemiologically linked index cases. Peak viral load did not differ by vaccination status or variant type [sic]. In Germany, the rate of symptomatic COVID-19 cases among the fully vaccinated (“breakthrough infections”) is reported weekly since 21. July 2021 and was 16.9% at that time among patients of 60 years and older [sic]. This proportion is increasing week by week and was 58.9% on 27. October 2021 (Figure 1) providing clear evidence of the increasing relevance of the fully vaccinated as a possible source of transmission. A similar situation was described for the UK. Between week 39 and 42, a total of 100.160 COVID-19 cases were reported among citizens of 60 years or older. 89.821 occurred among the fully vaccinated (89.7%), 3.395 among the unvaccinated (3.4%) [sic]. One week before, the COVID-19 case rate per 100.000 was higher among the subgroup of the vaccinated compared to the subgroup of the unvaccinated in all age groups of 30 years or more. In Israel a nosocomial outbreak was reported involving 16 healthcare workers, 23 exposed patients and two family members. The source was a fully vaccinated COVID-19 patient. The vaccination rate was 96.2% among all exposed individuals (151 healthcare workers and 97 patients). Fourteen fully vaccinated patients became severely ill or died, the two unvaccinated patients developed mild disease [sic]. *The US Centres for Disease Control and Prevention (CDC) identifies four of the top five counties with the highest percentage of fully vaccinated population (99.9–84.3%) as “high” transmission counties* [sic]. Many decisionmakers assume that the vaccinated can be excluded as a source of transmission. It appears to be grossly negligent to ignore the vaccinated population as a possible and relevant source of transmission when deciding about public health control measures [my emphasis].”



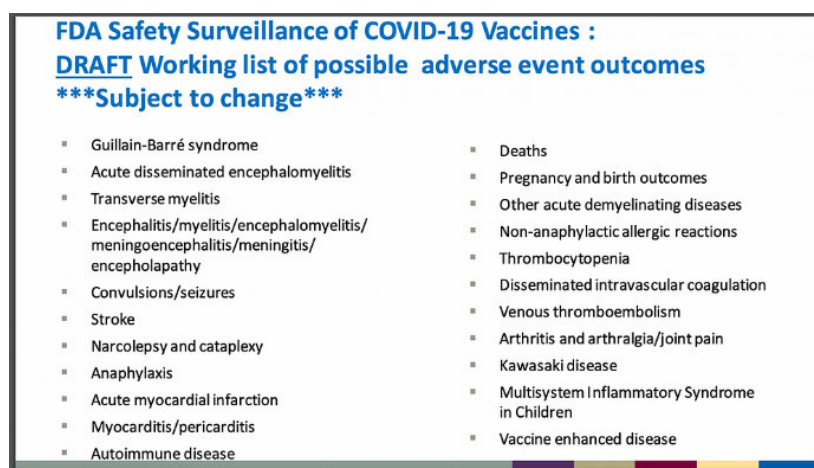
Based on these data alone, the logic behind the mass vaccination campaign and the mandates is totally invalidated. Again, as Kampf (2021) notes, the idea that unvaccinated individuals are somehow principally responsible for the continued circulation of SARS-CoV-2 has no evidentiary basis. It is blatantly false propaganda designed to scapegoat non-compliant portions of the population. Consider the following from Kampf (2021):

“There is increasing evidence that vaccinated individuals continue to have a relevant role in transmission. In Massachusetts, USA, a total of 469 new COVID-19 cases were detected during various events in July, 2021, and 346 (74%) of these cases were in people who were fully or partly vaccinated, 274 (79%) of whom were symptomatic. Cycle threshold values were similarly low between people who were fully vaccinated (median 22·8) and people who were unvaccinated, not fully vaccinated, or whose vaccination status was unknown (median 21·5), indicating a high viral load even among people who were fully vaccinated. In the USA, a total of 10 262 COVID-19 cases were reported in vaccinated people by April 30, 2021, of whom 2725 (26·6%) were asymptomatic, 995 (9·7%) were hospitalised, and 160 (1·6%) died. In Germany, 55·4% of symptomatic COVID-19 cases in patients aged 60 years or older were in fully vaccinated individuals, and this proportion is increasing each week. In Münster, Germany, new cases of COVID-19 occurred in at least 85 (22%) of 380 people who were fully vaccinated or who had recovered from COVID-19 and who attended a nightclub. People who are vaccinated have a lower risk of severe disease but are still a relevant part of the pandemic. *It is therefore wrong and dangerous to speak of a pandemic of the unvaccinated.* Historically, both the USA and Germany have engendered negative experiences by stigmatising parts of the population for their skin colour or religion. I call on high-level officials and scientists to stop the inappropriate stigmatisation of unvaccinated people, who include our patients, colleagues, and other fellow citizens, and to put extra effort into bringing society together.”

In sum, the vaccines are nowhere near effective enough to justify a global rollout, let alone *mandates*. The vaccines should probably be discontinued immediately for the vast majority of people given that they have been insufficiently tested and falsely advertised as highly effective.

3. Dangers for some demographics

The plethora of well documented AEs associated with vaccination against SARS-CoV-2 (especially for younger cohorts who are generally not at risk from SARS-CoV-2) should serve as the final nail in the coffin of the mass vaccination campaign. On October, 22, 2020, the Vaccines and Related Biological Products Advisory Committee (VRBPAC) of the FDA convened to discuss surveillance protocols regarding the current SARS-CoV-2 vaccine rollout. Topics included possible AEs that the VRBPAC anticipated would be associated with the currently available SARS-CoV-2 vaccines. Ultimately, the VRBPAC plays a significant role in vaccine approval by voting on whether vaccines should be approved and for whom. It is worth noting that, during their meeting, the group scanned over the following slide in roughly 2 seconds. It is also worth noting that, collectively, the members of the VRBPAC have earned (personally or on behalf of their institutions) hundreds of millions of dollars from the pharmaceutical industry and the Bill and Melinda Gates Foundation (The Defender, 2021). See the slide from their meeting below:



(Anderson, 2020)

To my knowledge, nearly all of the AEs listed in the slide have occurred, barring perhaps narcolepsy, cataplexy, as well as pregnancy and birth outcomes (which, of course, *cannot currently be assessed* due to the paucity of long-term data). In any case, it is important to note that data now show that the incidence rates for some severe AEs exceed the incidence rate for severe COVID-19 for at least some demographics (e.g., younger, immunocompetent cohorts). At the very least, and from a purely risk-benefit perspective, this should be enough to put an end to the mass vaccination campaign for those who are not at serious, immediate risk from COVID-19. Consider that Public Health Ontario (PHO) now notes that the incidence rate of myocarditis (i.e., inflammation of the myocardium) following vaccination with the NIH-Moderna vaccine for young men aged 18–24 is, according to official government data, 1 in 5,000 (Davidson, 2021). On their website, PHO note the following:

“As of November 21, 2021, there have been 537 reports of myocarditis or pericarditis following receipt of COVID-19 mRNA vaccines in Ontario. These reports have been identified through case-level review of all reported AEFIs. Of these, 149 (27.7%) were diagnosed with myocarditis and 242 (45.1%) were diagnosed with pericarditis. The remaining 146 (27.2%) were diagnosed with perimyocarditis (n=28), myopericarditis (n=115) and myocarditis/pericarditis (n=3).”

Additionally, for other vaccine combinations (e.g., first dose Pfizer-BioNTech, second dose NIH-Moderna), the incidence rate is upwards of 300 per million doses administered, or *1 in 3,000* (Buchan et al., 2021). Consider the following from Buchan et al. (2021):

“The highest reporting rate of myocarditis or pericarditis was observed in males aged 18-24 years following mRNA-1273 as the second dose, which (in our primary analysis) was 5.1 (95% CI 1.9-15.5) times higher than the rate following BNT162b2 as the second dose (299.5 vs. 59.2 per million doses, respectively). The second highest reporting rate was observed among males 12-17 following their second dose of BNT162b2 (97.3 per million [95% CI 60.3-148.8])

...

The rate among males aged 18-24 receiving two doses of mRNA-1273 were lower than those who received BNT162b2 followed by mRNA1273 (288.4 per million [95% CI 190.1-419.6] vs. 337.6 per million [95% CI 226.1-484.9], respectively). There were no reported events in males aged 18-24 years who received a first dose of mRNA-1273 followed by a second dose of BNT162b2; however fewer than 9,000 males in this age group received this schedule.”

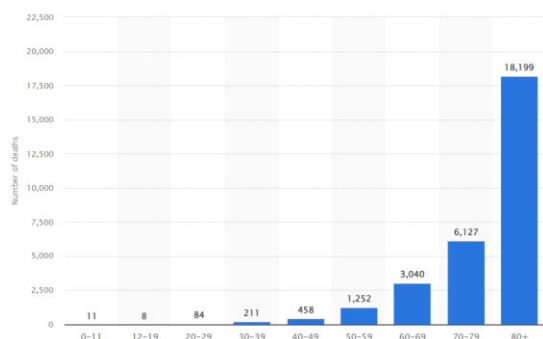
For males aged 18–24 who received two doses within a 30 day interval or less, the rate of vaccine-induced myocarditis was *777.2 per million doses administered*, i.e., *roughly one case for every 1300 doses administered* (Buchan et al., 2021). Observe:

Table 3. Reporting rate of myocarditis/pericarditis among males aged 18-24 years by vaccine products and inter-dose interval with dose 2 on or after June 1, 2021

Vaccine schedule	Reports (N)	Doses administered (N)	Rate (95% CI) per million doses
People with two doses			
BNT162b2-BNT162b2	11	235,819	46.6 (23.3 - 83.5)
Interval ≤30 days	2	21,160	94.5 (11.4 - 341.4)
Interval 31-55 days	8	124,235	64.4 (27.8 - 126.9)
Interval ≥56 days	1	90,424	11.1 (0.3 - 61.6)
mRNA-1273-mRNA-1273	27	93,616	288.4 (190.1 - 419.6)
Interval ≤30 days	4	10,623	376.5 (102.6 - 964.1)
Interval 31-55 days	20	60,352	331.4 (202.4 - 511.8)
Interval ≥56 days	3	22,641	132.5 (27.3 - 387.2)
mRNA-1273-BNT162b2	0	8,853	0 (0.0 - 416.7)
Interval ≤30 days	0	1,058	0.0 (0.0 - 3486.7)
Interval 31-55 days	0	5,402	0.0 (0.0 - 682.9)
Interval ≥56 days	0	2,393	0.0 (0.0 - 1541.5)
BNT162b2-mRNA-1273	29	85,893	337.6 (226.1 - 484.9)
Interval ≤30 days	6	7,720	777.2 (285.2 - 1691.6)
Interval 31-55 days	20	62,717	318.9 (194.8 - 492.5)
Interval ≥56 days	3	15,456	194.1 (40.0 - 567.2)

(Buchan et al., 2021)

Now, recall that as of November 26th, 2021, far less than 537 young men aged 18–24 have died *from or with* COVID-19:



(Elflein, 2021)

When interpreting these data, we must remember two crucial points. Firstly, COVID-19 mortality rates for all cohorts are confounded by the fact that regulatory public health agencies do not distinguish between (1) dying *from* symptomatic COVID-19, and (2) dying *after* testing positive for SARS-CoV-2 via RT-PCR. These are two very different things. Likewise, because of this failure to distinguish (1) from (2), hospitalization data can “greatly *overestimate* the true burden of COVID-19 disease in children [my emphasis]” (Zweig, 2021). Recall that Kushner et al. (2021) found the following in their study of pediatric COVID-19 hospitalizations in San Francisco:

“Of 117 hospitalizations, 46 (39.3%) were asymptomatic, 33 (28.2%) had mild to moderate disease, 9 (7.7%) had severe illness, and 15 (12.8%) had critical illness (weighted κ : 0.82). A total of 14 (12%) patients had multisystem inflammatory syndrome in children. A total of 53 (45%) admissions were categorized as unlikely to be caused by SARS-CoV-2 (κ : 0.78) [my emphasis].” (Kushner et al., 2021).

Hence, severe disease and COVID-associated deaths are overestimated by the official data. In any case, even with the flawed reporting of hospitalizations and deaths, the incidence rate of *just one* of many severe AEs associated with the SARS-CoV-2 vaccines eclipses the disease burden of severe COVID-19 for young, immunocompetent individuals! We must also consider the fact that AEs, especially in Canada, are significantly underreported. Therefore, the incidence rate of post-vaccination myocarditis for young men aged 18–24 is likely *even higher* than the official data convey. Consider the following conclusions drawn by Lazarus (2011) from an assessment of electronic vaccine injury reporting systems:

“Adverse events from drugs and vaccines are common, but underreported. Although 25% of ambulatory patients experience an adverse drug event, *less than 0.3% of all adverse drug events and 1-13% of serious events are reported to the Food and Drug Administration (FDA)*. Likewise, *fewer than 1% of vaccine adverse events are reported*. Low reporting rates preclude or slow the identification of “problem” drugs and vaccines that endanger public health. New surveillance methods for drug and vaccine adverse effects are needed. Barriers to reporting include a lack of clinician awareness, uncertainty about when and what to report, as well as the burdens of reporting: reporting is not part of clinicians’ usual workflow, takes time, and is duplicative. Proactive, spontaneous, automated adverse event reporting imbedded within EHRs and other information systems has the potential to speed the identification of problems with new drugs and more careful quantification of the risks of older drugs [my emphasis].”

Now, if we are generous in our estimates and assume that 537 reported cases of myocarditis in young males aged 18–24 constitute, say, 10% of all *actual* cases, then that would mean that there have been 5,037 cases of post-vaccination myocarditis in this demographic. That is assuming, of course, that the incidence rate remains constant for both the Pfizer-BioNTech and Moderna vaccines. This supposedly is not the case – PHO now recommends that males aged 18–24 receive the Pfizer-BioNTech vaccine instead (Davidson, 2021). That said, we should still be more than justified in estimating as high as 1 in 5,000 for the Pfizer-BioNTech vaccine as well given that “fewer than 1% of vaccine adverse events are reported” (Lazarus, 2011). The analysis conducted by Lazarus (2011) was conducted mainly to determine underreporting in the United States’ context, but I would argue that it applies equally well if not better to the Canadian context given that our AE reporting system is quite a bit more stringent in its criteria. We also need to consider that people are now encouraged to ‘mix and match’ vaccines, and that some combinations can, for young males, lead to even higher risks of developing myocarditis, i.e., between 1

in 1,300 to 1 in 3,000 (Buchan et al., 2021). Quite obviously, it follows that the vaccines are worse for young males than COVID-19.

Likewise, doctors in Ontario are clearly disincentivized from contraindicating any of the vaccines for their patients and, therefore, may be less likely to file AE reports for fear of being reprimanded. Indeed, there are examples of doctors being fired from their practice for filing too many AE reports (Carpay, 2021). Consider the following statement given by the College of Physicians and Surgeons of Ontario (CPSO) from April 30th, 2021:

“The College is aware and concerned about the increase of misinformation circulating on social media and other platforms regarding physicians who are publicly contradicting public health orders and recommendations. *Physicians hold a unique position of trust with the public and have a professional responsibility to not communicate [anti-vaccine](#), anti-masking, anti-distancing and anti-lockdown statements and/or promoting unsupported, unproven treatments for COVID-19.* Physicians must not make comments or provide advice that encourages the public to act contrary to public health orders and recommendations. Physicians who put the public at risk may face an investigation by the CPSO and disciplinary action, when warranted. When offering opinions, physicians must be guided by the law, regulatory standards, and the code of ethics and professional conduct. The information shared must not be misleading or deceptive and must be supported by available evidence and science [my emphasis].” (CPSO, 2021).

Given that the paragraph above contains a hyperlink to the picture below, I can only assume that physicians in Canada relay mainly the following information (and perhaps *only* the following information) about the SARS-COV-2 vaccines to their patients:

The facts about COVID-19 vaccines

Vaccination is one of the most effective ways to protect your health. Vaccines work with the body's natural defences to develop protection against a disease. COVID-19 vaccines provide instructions to your body's immune system to recognize and fight off the virus that causes COVID-19.

Free vaccines will be available to everyone who lives in Canada. Provinces and territories have developed detailed vaccination rollout plans for their residents.



Why get vaccinated for COVID-19?

Vaccines Work
Scientific and medical evidence show that vaccination can help protect you against COVID-19. Studies are also showing that vaccinated people may have less severe illness if they do become ill from COVID-19.

Vaccines Are Safe
Only vaccines that are proven to be **safe, effective** and of **high quality** are authorized for use in Canada. The COVID-19 vaccines have been rigorously tested during their development and then carefully reviewed by Health Canada.

The vaccines cannot give you COVID-19 because they don't contain the virus that causes it. The vaccines also cannot change your DNA.

mRNA vaccines provide instructions to your cells for how to make a coronavirus protein. This protein will trigger an immune response that will help to protect you against COVID-19.

Viral vector vaccines use a virus that's been made harmless to produce coronavirus proteins in your body without causing disease. Similar to mRNA vaccines, this protein will trigger an immune response that will help to protect you against COVID-19.

Continue to follow public health measures

COVID-19 vaccines are important tools to help us stop this pandemic. Right now, we still need to follow public health measures to reduce the spread of COVID-19 and save lives.

Federal, provincial and territorial governments will continue to assess the risk of COVID-19 spread in communities. Measures will be adjusted over time as more people are vaccinated, and we learn more about the science. Everyone is looking forward to a future when we can be together. Until then, we need to protect each other, especially those who are still vulnerable to severe disease from COVID-19.

Get the facts. Visit Canada.ca/covid-vaccine to learn more.




(Canada, 2020)

There really is not much going on here in the way of real information that could enable informed consent. Regardless, the important thing to note is that when we consider just one of many AEs (i.e., myocarditis), the SARS-CoV-2 vaccines ought to be ruled out for males aged 18–24. Likewise, when referencing the evidence from the clinical trials, most physicians likely do not mention that AEs are underreported in journal articles as well, and that most journal articles that more accurately capture the risks of AEs associated with pharmaceutical products go unpublished (Mayo-Wilson et al., 2019). Consider the following from Mayo-Wilson et al. (2019):

“We examined 52 public and nonpublic data sources about trials of quetiapine for bipolar depression using data from the Multiple Data Sources study. We extracted and compared information about systematic AEs (i.e., AEs assessed for all participants) in six prespecified domains: cardiovascular, cholesterol, endocrine, extrapyramidal symptoms, mania, and weight.

Eligible trials did not assess and report the same systematic AEs, and most results were not available in public sources. Overall, public sources reported 159 results, of which 92 of 159 (58%) included sufficient statistical information to calculate the treatment effect and its precision. Nonpublic sources reported 636 results; 630 of 636 (99%) reported sufficient statistical information.

Systematic AEs were defined and analyzed in many ways, which led to many numerical results. *Most systematic AEs were not mentioned in public sources.* To minimize bias, methods for defining and analyzing potential AEs should be prespecified in trial registers and protocols. All trial results should be publicly available so that stakeholders can compare benefits and AEs. Trials could report core sets of AEs to facilitate decision-making.”

Also, from Mayo-Wilson et al. (2019), consider the following:

“Trials of the same drug reported information about different AEs. *Information in public sources was inadequate for decision-making.* No public source reported all AEs, or all serious AEs, identified in nonpublic sources about the same trial. Of trials with only public sources, 2/15 (13%) gabapentin and 0/3 (0%) quetiapine trials grouped AEs by anatomic or physiological system.

...

Public sources contained little information about nonsystematic AEs, including serious AEs. Grouping might make nonsystematic AEs easier to detect; however, most public sources did not report grouped AEs. Standards are needed to improve the collection and reporting of nonsystematic AEs so that stakeholders can use trials to assess the balance of potential benefits and harms [my emphasis].”

Importantly, even with highly imperfect surveillance and reporting of AEs, the incidence rates of post-vaccination myocarditis, other cardiac adverse events (CAEs), and COVID-19 hospitalization for young men have been compared, and the results seem to clearly do not favour vaccination. Høeg et al. (2021) concluded the following in their database analysis of the US Vaccine Adverse Events Reporting System (VAERS):

“Post-vaccination CAE [cardiac adverse event] rate was highest in young boys aged 12-15 following dose two. For boys 12-17 without medical comorbidities, the likelihood of post vaccination dose two CAE is 162.2 and 94.0/million respectively. *This incidence exceeds their expected 120-day COVID-19 hospitalization rate at both moderate (August 21, 2021 rates) and high COVID-19 hospitalization incidence* [my emphasis].” (Høeg et al., 2021).

Indeed, concerns vaccine injuries would occur and exceed the risks of COVID-19 itself were raised much earlier on during the vaccine rollout. Israeli health officials reported the following earlier in 2021:

“The COVID-19 vaccine made by Pfizer and BioNTech appears to put young men at elevated risk of developing a heart muscle inflammation called myocarditis, researchers in Israel say. In a report submitted today to the Israeli Ministry of Health, *they conclude that between one in 3000 and one in 6000 men ages 16 to 24 who received the vaccine developed the rare condition*. But most cases were mild and resolved within a few weeks, which is typical for myocarditis.

...

In Israel, *which relied almost exclusively on the Pfizer-BioNTech vaccine in its early and fast vaccination drive*, the Ministry of Health in January assembled a panel led by Dror Mevorach, head of internal medicine at the Hadassah University Medical Center, to investigate the issue [my emphasis].” (Vogel, 2021).

Researchers have since produced similar findings. Chouchana et al. (2021) found that “the inflammatory heart reactions most frequently reported were myocarditis (1241, 55%) and pericarditis (851, 37%), *the majority requiring hospitalization* (n=796 (64%)). Overall, patients were young (median age 33 [21-54] years). The main age group was 18-29 years old (704, 31%), and mostly males (1555, 68%) [my emphasis].” For this demographic specifically, other cardiac events seem to be less rare than originally anticipated. Jain et al. (2021) found that “Sixty-three patients with a *mean age of 15.6 years* were included; 92% were male. All had received a messenger RNA vaccine and, except for one, presented after the second dose. Four patients had significant dysrhythmia; 14% had mild left ventricular dysfunction on echocardiography, which resolved on discharge; 88% met the diagnostic CMR Lake Louise criteria for myocarditis [my emphasis].” Mevorach et al. (2021) found that ““The incidence of myocarditis, although low, increased after the receipt of the BNT162b2 vaccine, particularly after the second dose among young male recipients.” Trogstad et al. (2021) concluded that, “In conjunction with other recently published case series and national vaccine safety surveillance data, this case series suggests a possible association between acute myocarditis and COVID-19 vaccination in young males and highlights a potential pattern in accompanying CMR [cardio vascular magnetic resonance] abnormalities.”

Unfortunately, the risks of myocarditis post-vaccination are not totally unique to young males. They may be much higher in young males, but consider that population data from the UK now show that for all individuals under 40 years of age, male or female, the risks of myocarditis were *higher* post-vaccination than post-infection by SARS-CoV-2 (Patone et al., 2021). Hence, *all* individuals aged 40 years or less are *likely at a greater risk of suffering negative health outcomes from the NIH-Moderna vaccine than from COVID-19*. Consider the following:

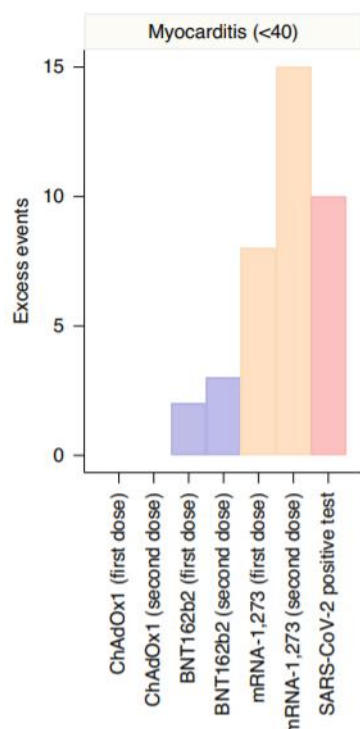


Fig. 2 | Number of excess events due to exposure per 1 million exposed

Again, these are the risks for both males and females aged 40 or less *combined*. For the former, the risks of myocarditis are higher, and for younger males the risks are *much higher* than they are from SARS-CoV-2 infection (Vinay Prasad MD MPH, 2021).

Beyond myocarditis (which is primarily caused by mRNA vaccines), there are myriad other AEs to consider. The Oxford-AstraZeneca vaccine was suspended in multiple countries due to the high incidence rates of various thrombotic events (Rogers, 2021). Among these events were incidences of Vaccine-Induced Thrombotic Thrombocytopenia (VITT) and Cerebral Venous Sinus Thrombosis (CVST), i.e., severe clotting disorders that often result in death (Science Table, 2021; Hughes, 2021). The Ontario Science Table (2021) note the following on their website:

“Published estimates of the risk of VITT currently range from 1 case per 26,500 to 1 case per 127,300 first doses of AstraZeneca/COVISHIELD administered. The risk of VITT in Canada has been estimated to be approximately *1 per 55,000 first doses*, but several possible cases are still under investigation. Early data from the United Kingdom suggests that the risk of VITT after second doses of AstraZeneca/COVISHIELD vaccines is likely lower than after first doses [my emphasis].”

Once again, an overall incidence rate for VITT of 1 case per 26,500 first doses will be based on *official government data* that could be underestimating the incidence rate by at least 90% (Lazarus, 2011). For CVST, Hughes (2021) notes the following:

““If we monitored this number of people in this age range [i.e., people aged 18–65] over 28 days, we would expect to see 0.3 cases of CVST [cerebral venous sinus thrombosis]. *We found a*

rate 20 times higher than that, which translates to 1 excess case in every 40,000 people vaccinated,” lead author Anton Pottegård, PhD, told *Medscape Medical News* [my emphasis].”

Crucially, both VITT and CVST are severe coagulopathies that often lead to death. For instance, Sánchez van Kammen et al. (2021) found that “In this cohort study of 116 patients with cerebral venous sinus thrombosis after SARS-CoV-2 vaccination ... [patients] often had intracerebral hemorrhage (68%) and concomitant thromboembolism (36%), and **47% died during hospitalization** [my emphasis].” Recall that the absolute risk of COVID-related mortality for individuals 18 and under can be as low as 0.0002% (Ledford, 2021). The *median* IFR for those aged 0–19 is 0.0027 (Axfors and Ioannidis, 2021). If we were to take the incidence rate of *just one* deadly AE, say, VITT occurring at a rate of 1 per 26,500 doses administered and generously compare it to the median COVID-19 IFR for those aged 0–19, then we are forced to draw the following conclusion:

VITT: $1 / 26,500 = 0.000037 * 100 = 0.0037\%$

MEDIAN COVID-19 IFR, 0–19: 0.0027%

$0.0037\% > 0.0027\%$

Therefore, I can only assume that if everyone aged 0–19 were vaccinated with the Oxford-AstraZeneca vaccine (that is, before it was discontinued indefinitely), then more of them would have died from vaccination than would have died from COVID-19. On this basis alone, vaccination with the Oxford-AstraZeneca vaccine should be ruled out, and yet, tens of millions of discarded doses from Canada alone are currently being donated to the global south, despite the obvious safety issues (Zimonjic, 2021). These rough risk-benefit analyses do not even consider the general lack of effectiveness at preventing COVID-19 that the vaccines have thus far demonstrated, and still, they fail on many fronts concerning safety alone! These analyses also do not consider the downstream effects of non-lethal VITT and other thrombotic events, such as disseminated intravascular coagulation, i.e., small clots forming in multiple vessels throughout the body (Stone et al., 2007). Needless to say, there is no logic in risking any of these known AEs (let alone the negative health outcomes that are presently unknown) if one is perfectly healthy and not at any appreciable risk from COVID-19.

Still, beyond cardiac events, coagulopathies, and other disorders of the circulatory system, the currently available SARS-CoV-2 vaccines have been linked with slightly less common neurological disorders and demyelinating diseases of the central nervous system (CNS). For instance, the Oxford-AstraZeneca vaccine is linked to cases of Acute Disseminated Encephalomyelitis (ADEM), a condition characterized by “demyelination in the brain and spinal cord” (Permezel et al., 2021). ‘Demyelination’ refers to the degradation of the myelin sheath surrounding nerve cells. The myelin sheath allows electrical impulses to be quickly transferred along the nerves. Crucially, if the myelin sheath is damaged, “these impulse slow down” (MedlinePlus, 2020). Hence, ADEM can lead to a number of ambulatory dysfunctions, such as vertigo, abdominal pain, nausea, seizures, fatigue, visual loss, dysmetria, gait instability, paresthesias, sphincter disturbance, and limb weakness (Permezel et al., 2021; Khayat-Khoei et al., 2021). In fact, there is a spectrum of neurological disorders associated with the currently available SARS-CoV-2 vaccines. Garg and Vimal (2021) note that “Another major neurological complication of concern is Bell’s palsy that was reported dominantly following mRNA vaccine administration. Acute transverse myelitis, acute disseminated encephalomyelitis, and acute demyelinating polyneuropathy are other unexpected neurological adverse events that occur as result of phenomenon of molecular mimicry.” Likewise,

Trimboli et al. (2021) note the following case report of Guillain-Barré syndrome (GBS), a form of severe paralytic neuropathy:

“Guillain-Barré syndrome (GBS) is the most common and severe acute paralytic neuropathy, with about 100,000 people developing the disorder every year worldwide. ... We describe a case of GBS after receiving the second dose of COVID-19 vaccine [BNT162b2—Pfizer®]. A 25-year-old woman without other comorbidities presented to our department on February 26th 2021 with a history of progressive lower limbs weakness and paresthesias for 4 days. She reported difficulty in walking and climbing stairs associated to paresthesias and numbness in the anterolateral region of the right thigh.”

Also, Perez-Lloret et al. (2021) note the following:

“Compared to BNT162b2, cerebral hemorrhagic or thrombotic events, headaches and migraines, Guillain-Barre syndrome and paresthesias, tremor and freezing, delirium, hallucinations, nervousness, poor sleep quality, and postural dizziness were more frequently reported with ChAdOx1. Reactions more frequently reported with BNT162b2 than ChAdOx1 were Bell’s palsy, facial paralysis, dysgeusia, anxiety, and presyncope or syncope.”

Woo et al. (2021) note the following:

“Within the US Vaccine Adverse Event Reporting System (VAERS), 130 cases of presumptive GBS were reported from February 2021 to July 2021. The overall estimated observed to expected rate ratio was 4.18, corresponding to an absolute rate increase of 6.36 per 100 000 person-years.

...

These findings suggest a potential small but statistically significant safety concern for Guillain-Barré syndrome following receipt of the Ad26.COV2.S vaccine but are considered preliminary pending analysis of medical records to establish a definitive diagnosis.”

The incidence rates for neurological disorders and various demyelinating diseases are, as far as I know, yet to be determined. That said, we do know that they are being underreported (Kelly Brown, 2021; Lazarus, 2011). We can also be sure that the cumulative incidence rate for all severe AEs associated with the Pfizer-BioNTech vaccine, i.e., those that are temporarily or permanently debilitating, require hospitalization, result in death, etc., is likely around 1 in 200 doses administered, which is higher for a lot of people than are the incidence rates of severe COVID-19 (Gøtzsche, 2021). When we consider that the NNV for all age cohorts sampled in the clinical trials is quite high, that the AAR is very low, and that the real-world effectiveness of the vaccines is diminishing precipitously every day, we must conclude that the currently available SARS-CoV-2 vaccines are simply not worth taking for most people.

Explanations for Adverse Events May Portend Long-Term Harmful Outcomes for Vaccine Recipients

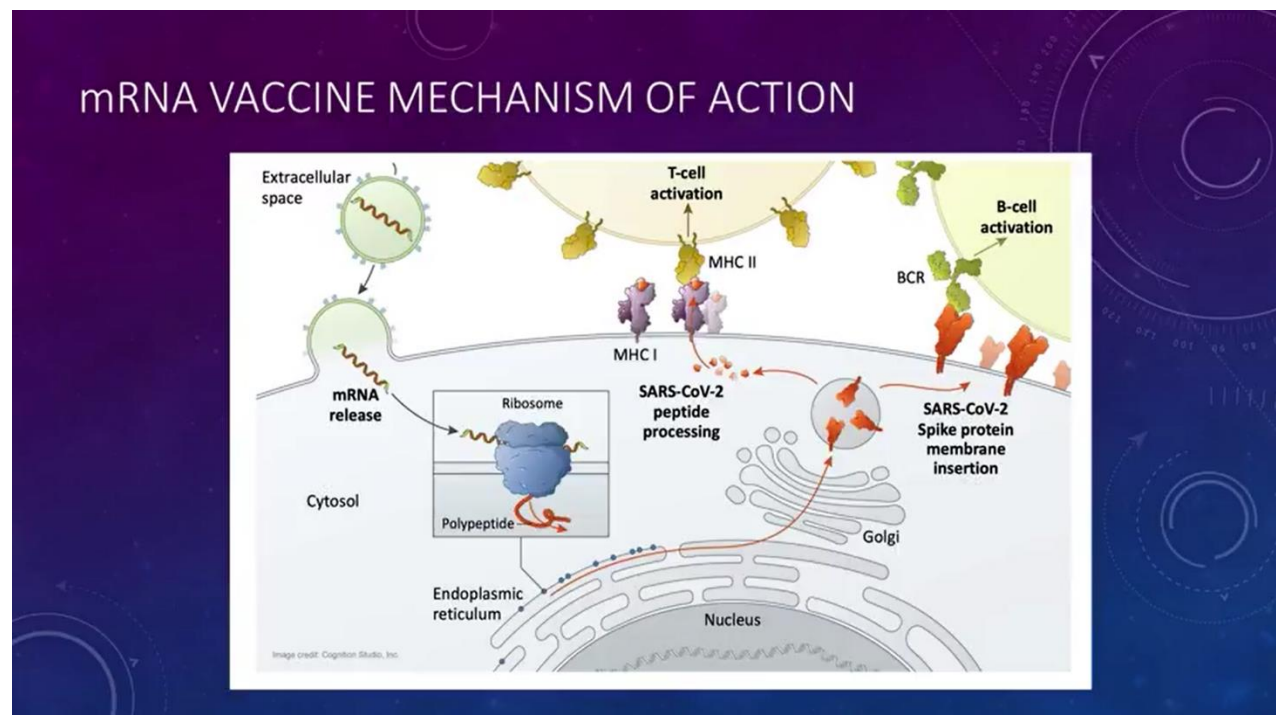
Briefly, I will touch on the fact that the known AEs should serve as a warning against mass vaccination. The known AEs indicate the potential for presently unknown long-term effects (let alone the effects of subsequent and/or *annual* booster vaccinations). Likewise, an *explanation* of the short-term AEs could serve as an indicator for which long-term effects are yet to come. Kostoff et al. (2021) note that the clinical trials were, obviously, not designed to detect long-term AEs. Consider the following:

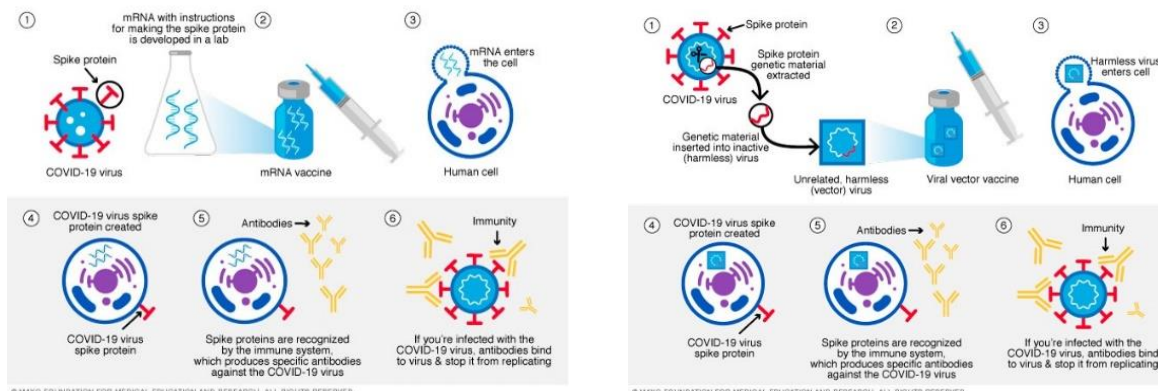
“During that symptom/disease development period, many biomarker early warning indicators tend to exhibit increasing abnormalities that reflect an increasing predisposition to the eventual symptom/disease. Thus, serious symptoms/diseases that ordinarily take long periods to develop would be expected to 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.

...

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 [e.g., cardiovascular dysregulation].” (Kostoff et al., 2021).

We simply do not know what will happen in the long-term. Damage leading to long-term harmful effects could be occurring subclinically in many people. Changes in various biomarkers would indicate whether this is happening, but it is still too soon to tell. Hence, as Kostoff et al. (2021) note, the clinical trials may only have captured very rare events, and the more common, systemic events that result from subclinical damage and/or changes to various biomarkers could take years to surface. Despite long-term data being non-existent, tentative *explanations* of AEs can go a long way in indicating which potential long-term complications can be expected. Many researchers are just starting to implicate the synthetic spike glycoproteins in the initiation of severe AEs, i.e., the target antigens that the mRNA and adenovirus vector vaccines cause human cells to synthesize and express. Firstly, consider the following representation of the mRNA and adenovirus vector vaccines’ mechanisms of action:





(Mayo Clinic Staff, 2020)

The mRNA vaccines contain lipid nanoparticles (LNPs) containing genetic material that can be translated into the synthetic spike glycoprotein, which is the target antigen against which the body's immune system is supposed to mount a protective immune response. For the mRNA vaccines, the delivery mechanism is, of course, messenger RNA contained in the LNPs that permeate the cell membrane. Then, once the mRNA is in the cell, ribosomes translate it into the spike glycoprotein, which is then expressed outwardly on the cell membrane. For the adenovirus vectors, the outcome is the same (i.e., spike protein expression), though, the delivery mechanism is an inactivated virus containing recombinant DNA that codes for the spike glycoprotein as opposed to LNPs containing mRNA. Immune cells then target the expressed spike proteins, thereby (hopefully) inducing protective immunity without causing too much damage in the process.

Crucially, earlier models of SARS-CoV-2 vaccine biodistribution did not anticipate that LNPs would be taken up by the deltoid's vasculature and then circulated widely beyond the injection site, thereby adversely affecting various tissue elsewhere in the body. They anticipated that the LNPs would stay localized in the injection site. However, researchers are now converging on explanations for severe AEs that single out widely biodistributed LNPs that lead to the production of spike proteins in extant regions of the body. Consider the following:

"Some of the mRNA in the vaccines may also be taken up by heart cells known as cardiomyocytes ... These cells may then produce the spike protein of the coronavirus, which could draw an antibody response against them, Dr. Schneider said. He said he has conducted lab tests and found that heart cells have taken up the Moderna vaccine and then expressed the spike protein [my emphasis]." (Loftus, 2021)

Essentially, when the LNPs circulate beyond the injection site and are deposited in extant regions of the body, they elicit strong immune responses against the otherwise healthy tissue in which the spike proteins are expressed. Immune responses, of course, kill cells. So, if the spike proteins expressed by cells that make up highly sensitive, non-regenerative tissue such as that of the heart muscle elicit an immune responses against said tissue, then many severe AEs could ensue, e.g., inflammatory diseases, like myocarditis. Some researchers have proposed 'solutions' to this problem. Consider this:

"...changing the vaccine injection site from deltoid to the vastus lateralis muscle of lateral mid-thigh may reduce the amount of vaccine lipid nanoparticles reaching the venous circulation due

to enhanced uptake by the dendritic cells and macrophages at the regional inguinal, iliac and para-aortic lymph nodes.” (Li et al., 2021).

What this finding suggests is that the vaccines do in fact enter the bloodstream through the deltoid’s vasculature, which has implications for the production of spike proteins in extant regions of the body, thereby resulting in the mounting of immune responses against delicate tissue. At this point, it is highly likely that the severe AEs are linked to wide LNP biodistribution and subsequent expression of the target antigen where it ideally should not be expressed. Vaccine-generated spike proteins have even been detected in the plasma of vaccine recipients (i.e., the largest part of the blood that carries water, salt, and enzymes all over the body), implying that the LNPs circulate beyond the injection site and that intact spike proteins can also circulate on their own (Ogata et al., 2021; Kostoff et al., 2021). This is significant, given that the vascular circulation of intact spike proteins has been linked to various complications, including (but not limited to) pulmonary hypertension (PAH) and other cardiac complications (Suzuki et al., 2021). Consider the following excerpt from Suzuki et al. (2021) on how the synthetic spike proteins can damage tissue:

“It is generally thought that the sole function of viral membrane fusion proteins is to allow the viruses to bind to the host cells for the purpose of viral entry into the cells, so that the genetic materials can be released and the viral replication and amplification can take place. However, *recent observations suggest that the SARS-CoV-2 spike protein can by itself trigger cell signaling that can lead to various biological processes*. It is reasonable to assume that *such events, in some cases, result in the pathogenesis of certain diseases*.

...

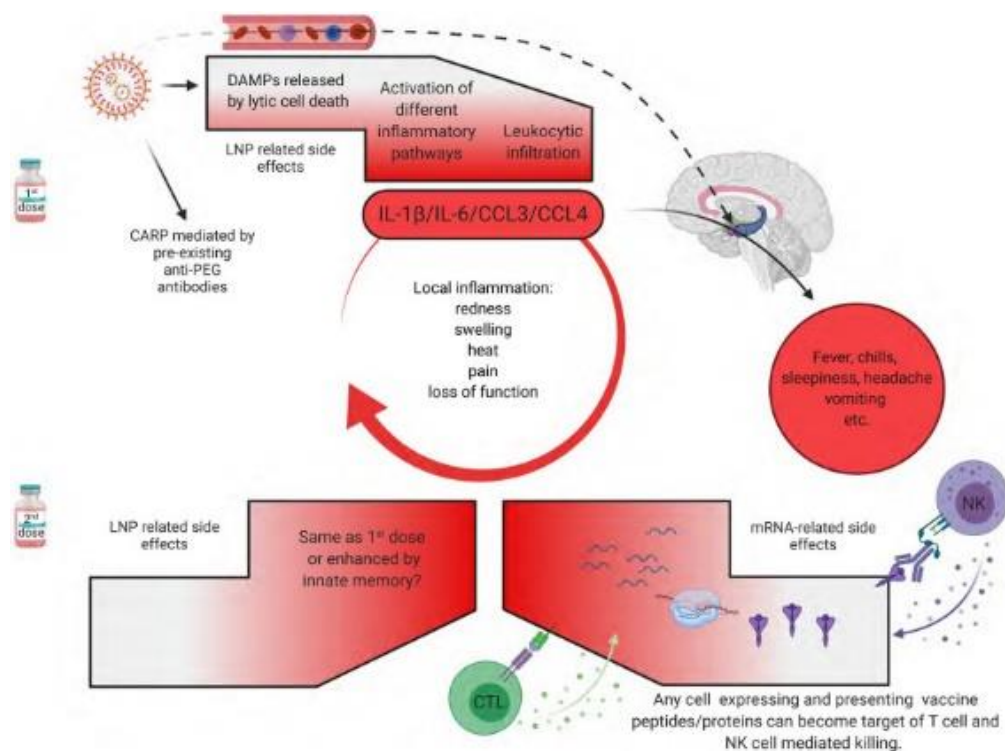
In addition to cardiovascular cells, other cells that express ACE2 [receptors] have the potential to be affected by the SARS-CoV-2 spike protein, which may cause adverse pathological events. Thus, it is important to consider the possibility that the SARS-CoV-2 spike protein produced by the new COVID-19 vaccines triggers cell signaling events that promote PAH, other cardiovascular complications, and/or complications in other tissues/organs in certain individuals [my emphasis].”

Ndeupen et al. (2021) have even sketched a highly informative model to explain all inflammatory and/or spike protein-induced AEs in vaccine recipients. Consider the following:

“In summary, the first vaccination’s side effects ... are likely associated with robust inflammation induced by the LNPs. In contrast, after the second vaccination, *side effects could be further exacerbated by immune responses targeting cells expressing the vaccine protein or its peptide derivatives*. Whether innate memory responses to LNPs also contribute to amplifying the side effects remains to be determined (Figure 4). Overall, the robust inflammatory milieu induced by LNPs, combined with presentation of the vaccine-derived peptides/protein outside of antigen-presenting cells, *might cause tissue damage and exacerbate side effects*. Since *self-antigen presentation in an inflammatory environment has been linked to autoimmune disease development*, this merits further investigation, albeit not detected here [my emphasis].

...

Figure 4.



“Potential mechanism of side effects.

...

Humans often experience more severe side effects with the second dose. Here we posit that might be due to multiple reasons. Firstly, innate immune memory against the LNPs might form after the first vaccination, and that could lead to even more robust inflammatory responses upon the second vaccination. Secondly, after the first vaccination adaptive immune responses are formed targeting the viral protein coded by the mRNA. As such, cells (shown as red shape) expressing the viral protein derived peptides or protein itself can become the target of CD8⁺ T or NK cell-mediated killing (ADCC), respectively. Since the LNPs could diffuse throughout the body and transfect any cell in their path with the mRNA, and the mRNA could also be further distributed through extracellular vesicles [sic], the target population could potentially be vast and diverse.” (Ndeupen et al., 2021).

In my view, this is the most likely explanation for severe AEs. It encompasses nearly all of the known AEs and attributes them to a common mechanism of action that is homogenous (e.g., spike proteins circulating throughout the vasculature) to all currently available mRNA and adenovirus vector SARS-CoV-2 vaccines. Likewise, Ndeupen et al. (2021) are correct to point out that “Since self-antigen presentation in an inflammatory environment has been linked to autoimmune disease development, this merits further investigation.” Long-term effects from vaccination against SARS-CoV-2 could, therefore, lead to the development of autoimmune disorders, and this could be further compounded by the introduction of annual booster doses. Unfortunately, we could see a future in which individuals are mandated to continuously receive treatments that have the paradoxical effect of diminishing their immunity to various other pathogens or subclinically damaging tissue such that degenerative disorders develop.

Interestingly, researchers recently proposed a drug candidate for treating what they term “Antibody Dependent Auto-Attack [ADAA]” (Wang et al., 2021). ADAA, the authors note, occurs when “anti-spike antibodies generated by COVID-19 or by the SARS-CoV-2 vaccines have a pathogenic effect through binding to ... cells and misleading immune responses to attack self-cells” (Wang et al., 2021). Evidently, if researchers are *already developing drugs* to treat autoimmune-type reactions from the SARS-CoV-2 vaccines, then this certainly merits concern from otherwise healthy people who are currently being mandated to receive the vaccines. Researchers currently developing new drugs must be anticipating serious problems.

Conclusion

In this paper, I have proven each of the following premises:

Premise 1: COVID-19 is not particularly deadly for most people given that (1) the risk of COVID-19 mortality is extremely age/morbidity-specific, and (2) deaths attributed to COVID-19 have been substantially overreported (at least in western countries).

Premise 2: Naturally acquired immunity against infection by SARS-CoV-2 is widespread and highly protective against symptomatic reinfection.

Premise 3: The currently available SARS-CoV-2 vaccines are insufficiently tested, relatively poor quality, and unsafe for at least some demographics.

Premise 4: If COVID-19 is not particularly deadly for most people, if naturally acquired immunity against infection by SARS-CoV-2 is widespread and highly protective, and if the currently available SARS-CoV-2 vaccines are insufficiently tested, relatively poor quality, and unsafe for at least some demographics, then mass vaccination (let alone mandatory vaccination) is unjustified.

Therefore, I conclude that mass vaccination against SARS-CoV-2 (let alone mandatory vaccination) is unjustified. The currently available SARS-CoV-2 vaccines should be discontinued for the vast majority of people who are not at risk from SARS-CoV-2 infection. The vaccines should be offered to elderly and/or immunocompromised people, but only if these people are given all of the facts beforehand. For instance, physicians may discuss the fact that the clinical trials demonstrate some efficacy against infection, but that they were not conducted long enough to determine whether the vaccines prevent hospitalization or death, or whether this efficacy lasts long-term. Likewise, they were not conducted long enough to determine which long-term effects could ensue. They have also been linked to a number of specific short-term serious AEs that are generally underreported, and so on, and so on. In this paper, I limited myself to discussing only the medical and/or public health-related arguments against mass vaccination. There are important sociopolitical implications of the vaccines and the concomitant ‘vaccine passport’ systems that are cropping up everywhere that deserve their own discussions. Also worthy of discussion are the various precedents that have been set, such as the fact that people can now be coerced by their governments into receiving novel, untested pharmaceutical products under the pretense of an ongoing ‘crisis’ while also being completely shut out of society if they refuse. More so than the medical and/or public health-related topics of discussion, I believe the sociopolitical topics warrant the most attention moving forward.

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