

# Notes on COVID

**The whole aim of practical politics is to keep the populace alarmed (and hence clamorous to be led to safety) by menacing it with an endless series of hobgoblins, all of them imaginary.**

H.L. MENCKEN ON COVID-19

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## Preface

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I could think of more interesting and edifying things to do than to review the utterly fraudulent science that is being peddled by the corrupt politicians and mainstream media, as well as by their equally corrupt academic hirelings, but I feel compelled to speak out against this misuse of science, which aims at taking away our freedom, or health, and our wealth.

You may be wondering what qualifies me for this undertaking. I am a medical doctor by training, with specialization and board certification in medical microbiology (all degrees from German institutions, no accreditation in Canada). Since the year 2001, I have been teaching and doing research in biochemistry at the University of Waterloo, Ontario, Canada. My background therefore qualifies me to speak to both the scientific and the clinical aspects of viral infections. I want to make it clear, however, that I am not speaking on behalf of my university, but as a private citizen.

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# Chapter 1

## The COVID virus was made in the lab

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In order to better understand what this entire fake pandemic is all about, we can start by looking at the two competing narratives as to the origin of the SARS-CoV-2 virus. The story that is promoted by the mainstream media, including the mainstream peer-reviewed scientific journals, is that the virus originated in bats or some other wild animal species and somehow hopped over to humans. Other people maintain that the genome of the virus shows clear evidence of artificial tampering. Which side is right? Let's find out.

### 1.1 Introduction

#### 1.1.1 A graphical abstract



In recent years, scientific journals have embraced the idea of the “graphical abstract,” requiring their authors to capture the main idea of each article in a single picture. While this is usually difficult to do and often unsuccessful, it is quite easy in this particular case.

If you have better things to do than to read this chapter and simply want the “takeaway message,” let me introduce the Wolpertinger—a mythical creature from Bavaria. You may think of SARS-CoV-2 as the microscopic counterpart of the Wolpertinger. If you can bring yourself to accept that the Wolpertinger is real and natural, you should have no difficulty believing the same of SARS-CoV-2, too.

### 1.1.2 Does SARS-CoV-2 even exist?

- Multiple studies report having grown the virus in cell culture and identified the virus in these cultures with PCR or antibodies
- In spring 2020, there was indeed a surge of viral infections and, in some jurisdictions, of deaths in nursing homes
- Also in spring 2020, a wave of a severe acute inflammatory disease (Kawasaki syndrome) was observed in children

An overview of studies reporting the cultivation of SARS-CoV-2 is provided by Jefferson et al. [1]. The regional differences in mortality are examined by Rancourt [2]. Reports of increased incidence of the normally rare Kawasaki syndrome, a potentially severe generalized inflammation most common in young children, are reviewed by Abrams et al. [3]. A high percentage of these cases did have positive antibody tests for SARS-CoV-2.

These findings consistently indicate that SARS-CoV-2 exists and infects humans. This does not imply the virus is responsible for all acute disease in people infected by it, or that the results of indirect tests (PCR/antibodies) are always reliable. We can assume that during the flu season 2020/21, most disease will be caused by viruses other than SARS-CoV-2, even among those with flu-like symptoms *and* a positive COVID test.

### 1.1.3 The main source for this presentation: Li-Meng Yan

Li-Meng Yan has published her analyses in two preprints [4, 5]. She states that both reports were submitted for publication to a peer-reviewed journal but were rejected. However, the quality of these publications is sound. The reviews that motivated the rejection can be found online [6]; they are superficial and ignore much of the evidence which Yan and her co-authors present to support their conclusions.

My impression of Yan’s reports is that some “intelligence community” types lent a hand in crafting them. Such people can certainly be expected to take a lively interest in her work. It is also clear that Yan must be enjoying some protection. She gives as her affiliation the Rule of Law Society in New York, which is linked to Steve Bannon, and therefore to the Trump government.



- MD/PhD virologist from Hong Kong University—recently fled to U.S., apparently now under Steve Bannon’s protection
- I suspect that her back story contains some inaccuracies, but that is unrelated to scientific substance of her work
- Her papers have not been published by peer-reviewed journals—but they are scientifically sound regardless

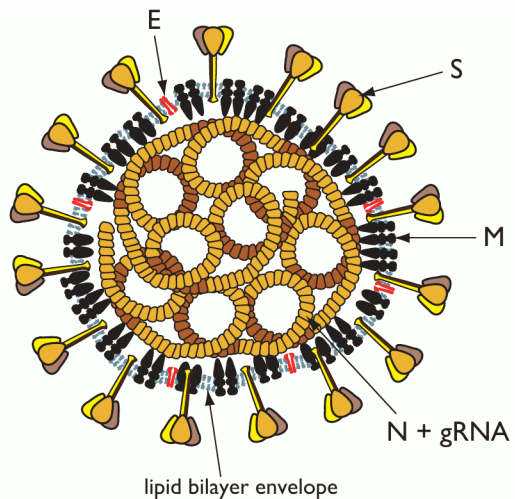
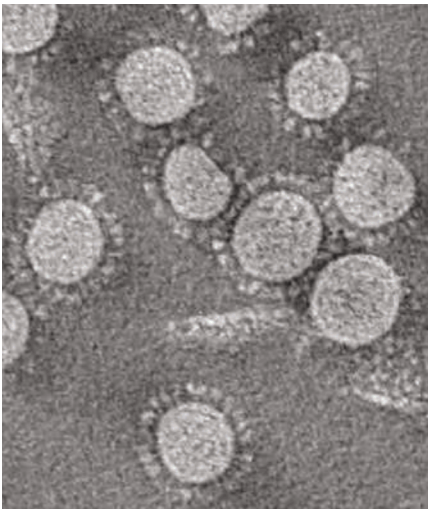


We here simply take note of these facts; they will not prejudice the following analysis in any way. The science can be evaluated without reference to the back story.

## 1.2 Background on coronavirus biology

Before we dig into the details of SARS-CoV-2 and its natural or unnatural origins, we will review some basic principles and facts pertaining to the virus family it belongs to, the coronaviruses.

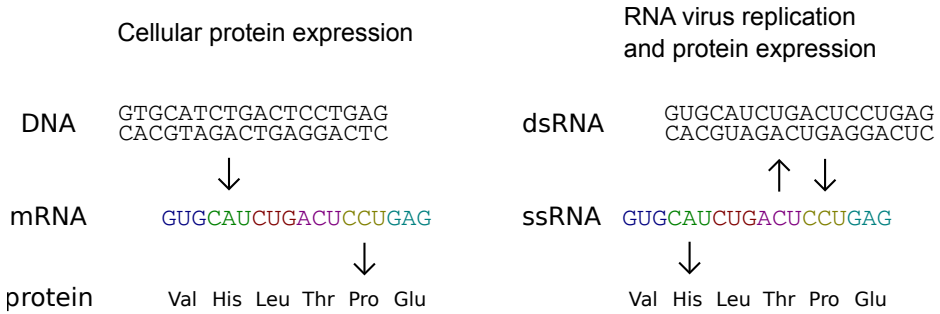
### 1.2.1 Structure of coronavirus particles



This picture (taken from [7, p. 828]) shows the electron-microscopic appearance of coronaviruses (left) and a schematic of the particle structure. Each of the prominent, club-shaped spikes (S) consists of three intertwined protein molecules; the entire complex is embedded in the virus envelope. The envelope is a lipid membrane, similar to and derived from the cell membrane of the host cell which produced the virus particle. Also embedded in this membrane are at least two more proteins (M

and E). The RNA genome of the virus, packaged into multiple copies of the N protein, is located in the interior of the particle.

### 1.2.2 Coronavirus RNA serves a dual role



DNA (deoxyribonucleic acid) and RNA (ribonucleic acid) are very similar, but the subtle difference between them means that DNA is more chemically stable, and therefore more suitable for storing genetic information with high fidelity. Accordingly, the cells of the human body store and transmit their genetic information in the form of double-stranded DNA. In contrast, coronaviruses such as SARS-CoV-2 store theirs in the form of single-stranded RNA (ssRNA). This genomic RNA also directs protein synthesis, much like the messenger-RNA (mRNA) does in our own cells. Amplification of the ssRNA involves a double-stranded intermediate (dsRNA).

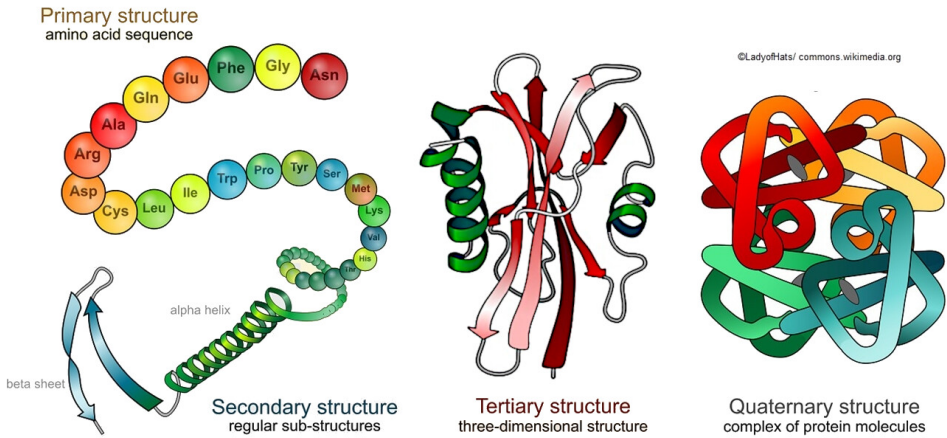
We will note here that it is possible *in vitro* to use PCR (polymerase chain reaction) to create a double-stranded DNA copy of a single-stranded RNA virus genome. From such a copy, the cellular RNA polymerase, whose regular job it is to transcribe cellular DNA into mRNA, can make single-stranded RNA copies again, which will then start replicating like a virus. This also means that we can manipulate the DNA copy of the virus using the entire tool set of recombinant DNA technology, and then leave it to some cells in a petri dish to turn such altered versions of the virus genome into live virus particles.

### 1.2.3 Proteins fold into complex structures

We saw that coronaviruses contain both proteins and RNA. Proteins are the most fascinating of all biological macromolecules. While nucleic acids consist of only four different building blocks, which furthermore have rather similar physico-chemical properties, proteins comprise twenty different building blocks—the amino acids. Furthermore, the amino acids have rather diverse chemical properties, some being positively charged, others negatively; those which are uncharged may be polar or apolar, bulky or small, and so on. This gives proteins a much greater scope of chemical variability than we find with nucleic acids.

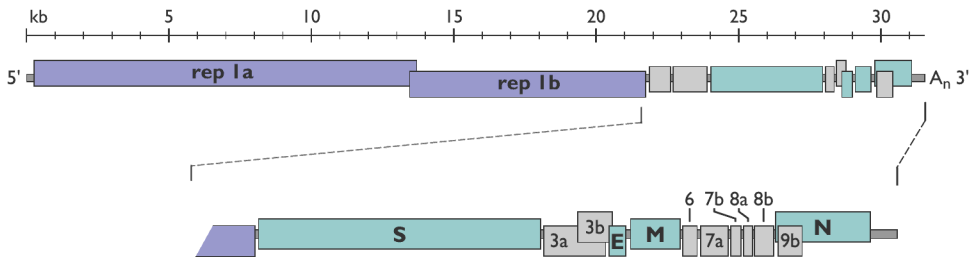
Within each protein molecule, all amino acids are connected like pearls on a string. However, this string will soon fold back upon itself, driven by mutual

interactions between all these different amino acids. Within the resulting folded structure, we can distinguish several levels of organization. It is this folded structure that determines the function of a protein molecule. There seems to be no limit to Nature’s ingenuity in the use of proteins—they do literally every job there is to do, from transporting and degrading foodstuffs over muscle contraction to detecting light, sound, or smell in the sensory cells of the eye, ear, and nose.



Coronaviruses, too, possess a number of proteins that fulfill different roles—some constitute the virus particle, whereas others exist only in the host cell and serve in various roles during intracellular virus multiplication. The amino acid sequences of all of these viral proteins are encoded on the single viral RNA molecule.

#### 1.2.4 Map of a coronavirus RNA genome



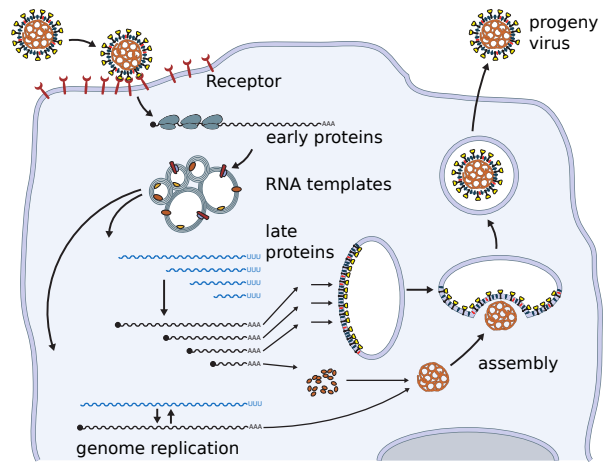
This map, which pertains to the SARS virus, is only meant to illustrate that the single RNA molecule contains the genes for all proteins that are needed to replicate the RNA (purple), to form the virus particle (green), and for several auxiliary functions (gray).

In the following, we will focus on the spike protein of the virus. The gene which encodes this protein is labeled with ‘S’. Graphic adapted from [7].



### 1.2.5 The coronavirus replication cycle

- virus particle binds to cellular receptors
- entry and uncoating of the RNA genome
- synthesis of early proteins for use in subsequent stages
- synthesis of late proteins and of RNA copies
- assembly of structural proteins and RNA genome into progeny virus
- exit of progeny virus

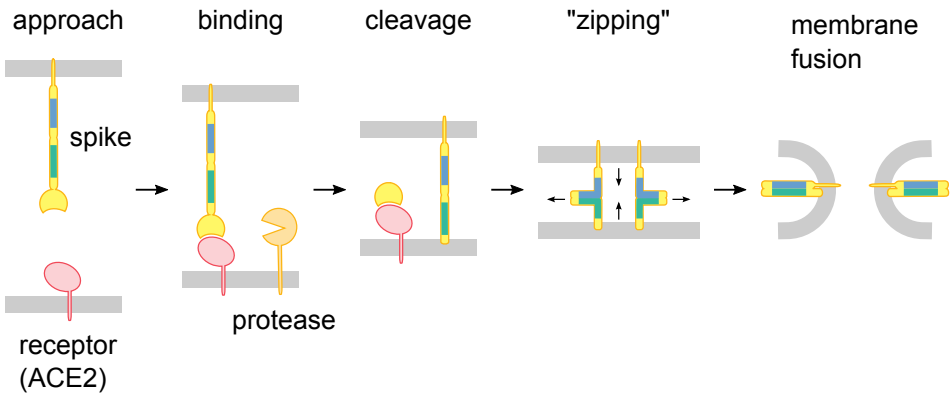


A crucial first step in the replication of any virus is its attachment to the host cell. This involves the mutual recognition between the virus spike protein and a receptor protein on the host cell membrane. In the case of SARS-CoV-2 and related coronavirus strains, the cellular protein is angiotensin-converting enzyme 2 (ACE2). This protein does of course not just exist for binding these viruses; instead, it serves an important role in human physiology, and the viruses have evolved to use it for their own ends because they can rely on its presence.

The above scheme (slightly modified from [7]) glosses over the question how the viral RNA gains entry into the host cell. This step involves the fusion of the viral envelope to the host cell membrane; this, too, is effected by the viral spike protein and will be considered below. The subsequent steps of viral replication are quite interesting as well, but for the purpose of this presentation we will focus on the spike protein, because it has a key role in host cell specificity, and most of the evidence of laboratory tampering pertains to this protein.

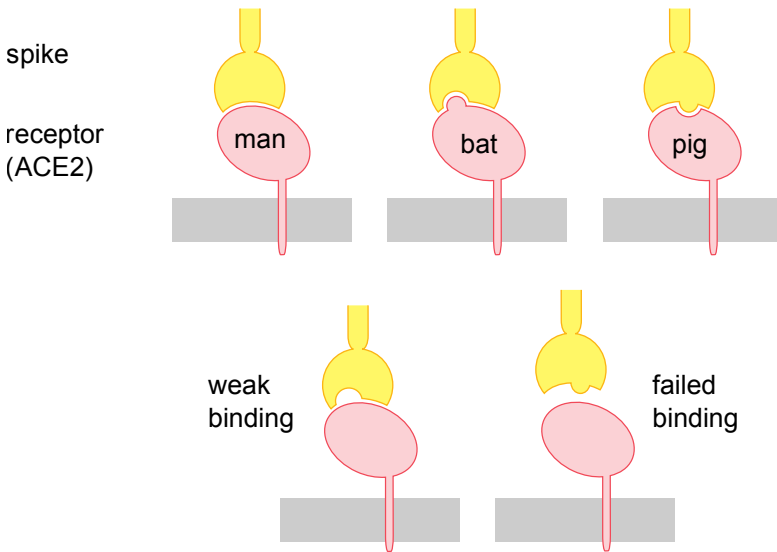
### 1.2.6 The S (spike) protein mediates binding to and fusion with the host cell

We had noted that the spike protein mediates not only binding but also the fusion of the viral envelope to the cell membrane. This second step is triggered when a host cell *protease*, that is, a protein which cleaves other proteins, does just this to the spike protein. The truncated spike protein then ‘harpoons’ the host cell membrane, and it also starts zipping up against itself, which forces the two membranes together and finally causes them to fuse. The fusion pore thus created permits the viral RNA to enter the cell.



**1.2.7 The spike proteins of different virus strains are adapted to the receptors in different animal hosts**

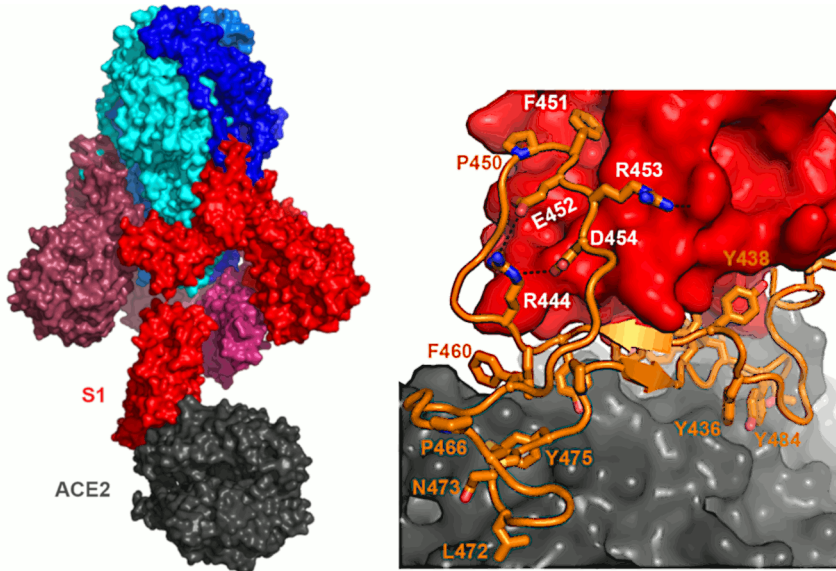
The mutual recognition between the viral spike protein and the host cell receptor crucially determines the host range of a given virus. Each coronavirus strain is optimally adapted to one or a few animal species. Sometimes, a virus strain may succeed in jumping to a new animal species; if so, its spike protein will be under intense selective pressure to improve its binding affinity for the cellular receptor protein in this species.



A rapid evolution towards improved receptor binding was indeed observed with the original SARS virus in the early 2000s. In contrast, with SARS-CoV-2, the affinity for the human receptor was already very high when the first strains were isolated for human patients. That means one of two things—either that the receptor had been optimized in the lab in just such a manner, or that the virus had already been

circulating for a considerable period of time in the human population. Of course, these two are not mutually exclusive—both might apply.

### 1.2.8 The actual structure of the spike and the receptor proteins



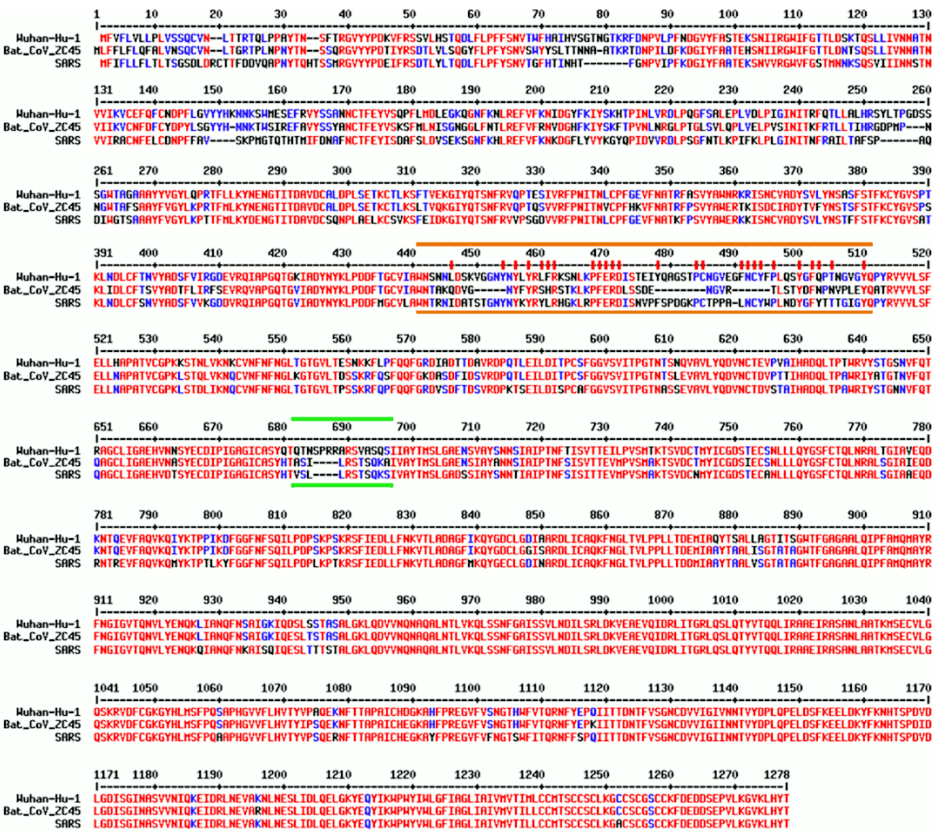
This slide (taken from [4]) shows the experimentally determined structure of the spike protein of the original SARS virus bound to the ACE2 receptor. All the colorful parts belong to the spike, whereas ACE2 is shown in gray. The important point here is illustrated in the right panel: the part of the spike protein that is displayed as an orange ‘sausage’ with some additional decoration, which makes direct contact with the receptor, dominates the interaction; any changes that may be required for adapting the spike to a new host receptor tend to cluster in this relatively small part of the molecule.

Below, we will refer to this part of the spike protein as the *receptor-binding domain* (RBD).

## 1.3 Analysis of the SARS-CoV-2 spike protein

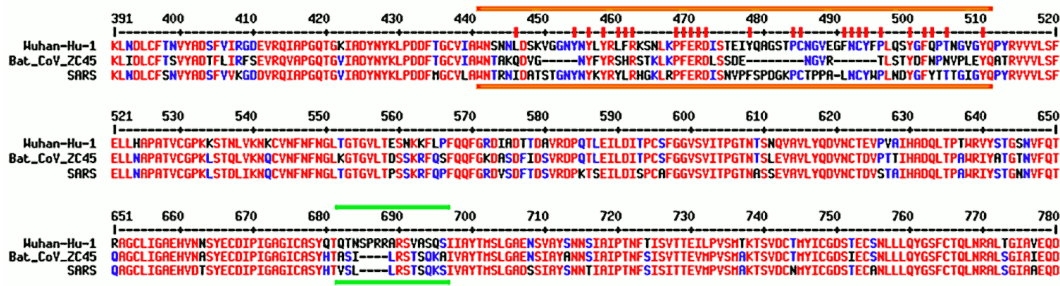
### 1.3.1 Comparison of the spike protein sequences from three different coronavirus strains (1)

This slide (modified from [4]) aligns the sequences of three coronavirus strains. They include a SARS-CoV-2 strain from Wuhan, a related virus (ZC45) which Yan et al. [4] name as the most likely starting point for the creation of SARS-CoV-2, and the original SARS virus. In earlier slides, each amino acid had been identified with three letters, whereas here every amino acid gets only one letter. All amino acids shown in red are shared by all virus strains; those in blue are shared by two, and those in black are not shared by any.



We note that more deviations (blue or black) occur near its end. In their analysis, Yan et al. [4] focus on the two variable stretches highlighted with orange and green bars, respectively.

### 1.3.2 Comparison of the spike protein sequences from three different coronavirus strains (2)

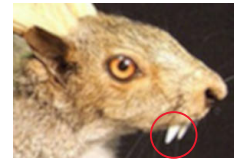
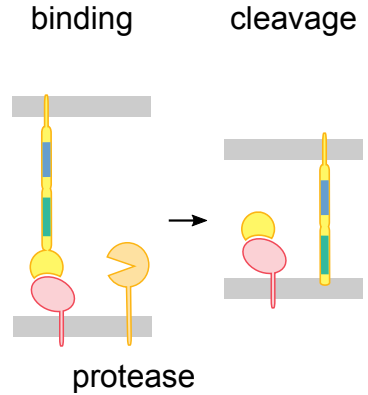


This slide shows the part of the protein sequence which comprises the two sites in question. The stretch highlighted by the orange bars corresponds to the receptor-binding domain shown in the folded 3D structure in slide 1.2.8. The green bars highlight a site at which SARS-CoV-2 contains a unique insertion of four amino acids. This insertion creates a cleavage site for the protease furin, and it constitutes the first piece of evidence of laboratory tampering with the SARS-CoV-2 spike protein.

### 1.3.3 SARS-CoV-2 is the only family member with a furin cleavage site

Human SARS-CoV BJ01	655	-	GICASYHTVSLI	---	RSTS	-	670
Human SARS-CoV CUHK-W1	655	-	GICASYHTVSLI	---	RSTS	-	670
Human SARS-CoV Tor2	659	-	GICASYHTVSLI	---	RSTS	-	670
Human SARS-CoV Frankfurt-1	655	-	GICASYHTVSLI	---	RSTS	-	670
Human SARS-CoV Urbani	655	-	GICASYHTVSLI	---	RSTS	-	670
Civet SARS-CoV civet020	655	-	GICASYHTVSLI	---	RSTS	-	670
Civet SARS-CoV sz16	655	-	GICASYHTVSLI	---	RSTS	-	670
Raccoon dog SARS-CoV A030	655	-	GICASYHTVSLI	---	RSTS	-	670
<b>SARS-CoV-2</b>	<b>669</b>	-	<b>GICASYQTQNSRRRNSVA</b>	-	<b>688</b>	-	<b>688</b>
Panolin CoV MP789	n/a	-	GICASYQTQNS	---	RSVS	-	n/a
Bat SARS-CoV RaTG13	669	-	GICASYQTQNS	---	RSVA	-	684
Bat SARS-CoV LYRa11	659	-	GICASYHTASLI	---	RNTD	-	674
Bat SARS-CoV LYRa3	659	-	GICASYHTASLI	---	RNTG	-	674
Bat SARS-CoV RsSHC014	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV Rs4084	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV WIV1	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV Rs3367	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV Rs7327	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV Rs9401	656	-	GICASYHTVSLI	---	RSTS	-	671
Bat SARS-CoV Ra4231	655	-	GICASYHTASLI	---	RSTG	-	670
Bat SARS-CoV WIV16	655	-	GICASYHTVSLI	---	RSTS	-	670
Bat SARS-CoV Rs4874	655	-	GICASYHTVSLI	---	RSTS	-	670
Bat SARS-CoV ZC45	646	-	GICASYHTASLI	---	RSTS	-	661
Bat SARS-CoV ZXC25	645	-	GICASYHTASLI	---	RSTG	-	660
Bat SARS-CoV RF4092	636	-	GICASYHTASLI	---	RSTG	-	651
Bat SARS-CoV RF/JL2012	636	-	GICASYHTASLI	---	RSTG	-	651
Bat SARS-CoV JTMCI5	636	-	GICASYHTASLI	---	RSTG	-	651
Bat SARS-CoV 16B0133	636	-	GICASYHTASLI	---	RSTG	-	651
Bat SARS-CoV B15-21	636	-	GICASYHTASLI	---	RSTG	-	651
Bat SARS-CoV YK213	634	-	GICASYHTASLI	---	RSVG	-	649
Bat SARS-CoV Anlong-103	633	-	GICASYHTASLI	---	RSVG	-	648
Bat SARS-CoV Rp/Shaanxi2011	640	-	GICASYHTASLI	---	RSTG	-	655
Bat SARS-CoV Ra/HuB2013	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV YMLF/34C	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV YN2/31C	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV RE1	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV 273	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV RF/SX2013	639	-	GICASYHTASLI	---	RSTG	-	654
Bat SARS-CoV RF/HuB2013	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV Cp/Xunnan2011	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV Ra672	641	-	GICASYHTASLI	---	RSVG	-	656
Bat SARS-CoV Rs4255	641	-	GICASYHTASLI	---	RSVG	-	656
Bat SARS-CoV 4081	641	-	GICASYHTASLI	---	RSVG	-	656
Bat SARS-CoV Rm1	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV 279	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV Ra/GX2013	642	-	GICASYHTASLI	---	RSTG	-	657
Bat SARS-CoV Rs806	641	-	GICASYHTASLI	---	RSTG	-	656
Bat SARS-CoV HKU3-1	642	-	GICASYHTASLI	---	RSTG	-	657
Bat SARS-CoV Longquan-140	642	-	GICASYHTASLI	---	RSTG	-	657
Bat SARS-CoV Rp3	641	-	GICASYHTASLI	---	RSVG	-	656
Bat SARS-CoV Ra4247	642	-	GICASYHTASLI	---	RSVG	-	657
Bat SARS-CoV Rs4237	641	-	GICASYHTASLI	---	RSVG	-	656
Bat SARS-CoV As6526	641	-	GICASYHTASLI	---	RSVG	-	656

SARS-COV 2



We had already discussed that cleavage by host cell proteases is needed to activate the spike protein for the subsequent fusion between virus and cell membranes (see slide 1.2.6). Inserting a cleavage site for another protease—in this case, furin—into the spike protein will potentially increase the number of cell types susceptible to the virus, because furin is found on the surfaces of many different cell types.

While furin cleavage sites similar to that of the SARS-CoV-2 spike protein do occur in some distantly related coronavirus strains [7], they do not occur among the SARS family (see sequence alignment on the left) or other virus strains closely related to SARS-CoV-2. The absence of a furin site in those relatives means two things:

1. Nature really does not want this site to be there; and if we introduce it, then we must expect that it will be lost due to natural selection as the virus propagates in cell culture or in animal or human hosts. In this connection, Yan et al. note that the DNA sequence for this furin site has been designed such that it can be cleaved using the restriction endonuclease *FauI*,<sup>1</sup> which makes it easy to confirm the continued presence of this site in progeny virus.
2. There is no plausible pathway for SARS-CoV-2 to have acquired this site the natural way, which means through exchange of genetic material with other virus strains.<sup>2</sup> Such genetic exchange will occur efficiently only between strains which

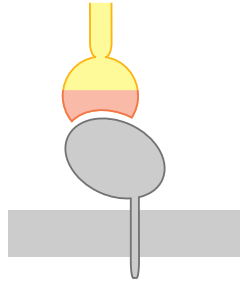
<sup>1</sup>No connection to Anthony Fauci.

<sup>2</sup>For this to occur, two virus strains must infect the same host cell; exchange of RNA segments can then occur during RNA replication.



already share a high degree of similarity—but the viruses similar to SARS-CoV-2 do not contain a furin site and therefore could not have supplied it.

### 1.3.4 The receptor-binding domain of SARS-CoV-2 retains key features from the original SARS virus

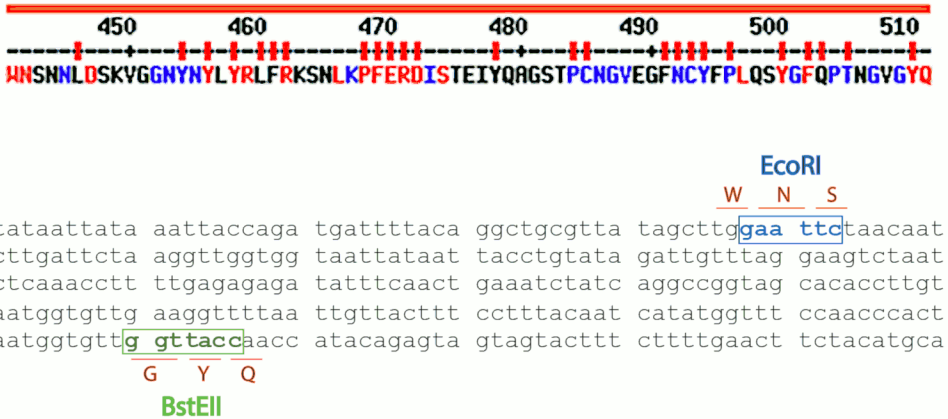


We now return to the receptor binding domain (RBD). We see that within this domain the three viruses show considerable variation, but note that SARS-CoV-2 and SARS have the exact same number of amino acids. In contrast, ZC45, whose genome on the whole has greater sequence similarity to SARS-CoV-2 than does SARS, shows several gaps in the RBD sequence. Therefore, even though ZC45 or a close relative of it seems to have provided most of the genome for SARS-CoV-2, the receptor binding domain must have come from somewhere else.

The red markers in the line above the three sequences indicate amino acid positions that are understood to be crucial for the interaction of the spike protein with the human ACE2 receptor. If we focus on SARS and SARS-CoV-2 only, we see that most of the positions flagged by red markers are occupied by the same amino acids. In positions where SARS and SARS-CoV-2 diverge, the two amino acids in question are chemically similar.

Yan et al. [4] suggest that the SARS-CoV-2 RBD was derived from the SARS RBD through extensive experimental variation: as many amino acid positions as possible were changed without compromising binding to the human receptor protein, so as to disguise this reuse.

### 1.3.5 The DNA sequence that encodes the receptor-binding domain is book-ended by two convenient restriction sites



The origin of the RBD suggested by Yan et al. would have involved the experimental testing, probably mostly in cell culture, of a large number of recombinant spike protein variants. In this context, the observation depicted in this slide is significant: at the beginning and at the end of the DNA sequence encoding the RBD, we find recognition sites for the two restriction enzymes *EcoRI* and *BstEII*, respectively. These sites would have made it easy to swap out the DNA segment between the two sites, that is, the RBD, while leaving the remainder of the spike protein unchanged. Experiments of exactly this kind have been reported by researchers from Wuhan before [8].

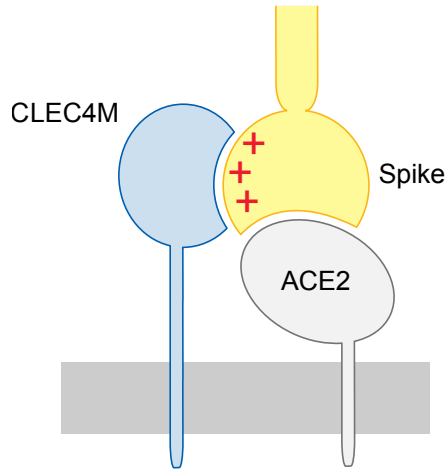
The probabilities of these two sites occurring in their exact spots is  $1/3000$  for *EcoRI* and  $1/8000$  for *BstEII*, which means that the combined probability is 1 in 24 million.<sup>3</sup>

Yan et al. submitted their study for peer review. The reviews they received are publicly available. Reviewer Adam Lauring of the University of Michigan criticizes Yan for attaching “*inordinate significance to a restriction enzyme site near the receptor binding domain. They consider it something of a smoking gun as it will allow for sub cloning of receptor binding domains during the engineering process. This site is a 6 nucleotide recognition sequence and would occur by chance once every 4096 bases in a genome sequence.*” The other reviewers don’t comment specifically.

Lauring’s estimate of such a site occurring once every 4096 bases assumes that all 4 bases (building blocks) occur with equal frequency, which is incorrect; but his estimated probability is not too far off the actual value. Much more gravely, however, Lauring ignores that *two* such sites occur here in combination. While Lauring’s omission *might* be due to incompetence, this is not the most likely explanation.

<sup>3</sup>In my video presentation, I estimated each site’s probability to be  $1/2000$ ; the values given here take into account the exact nucleotide composition of the viral genome and therefore are more accurate. In calculating the probability for the *BstEII* site, we must consider that only 6 out of the 7 comprised nucleotides (bases) are specifically recognized by the enzyme; the fourth position may be occupied by any nucleotide.

### 1.3.6 The SARS-CoV-2 spike protein contains mutations that recruit a second cellular receptor



In addition to the unique furin recognition site and the peculiarities of the RBD, the SARS-CoV-2 spike protein contains a series of changes that enable it to recruit a second cellular protein (known as CLEC4M or also as CD209) as a receptor for the initial host cell attachment. These changes are described in detail by Sørensen et al. [9, 10]. Collectively, they endow the spike protein with significantly increased positive electric charge, which attracts the negatively charged CLEC4M molecule.

The recruitment of CLEC4M for cell binding and entry, first suggested by Sørensen et al., has since been confirmed experimentally [11]. As with the addition of the furin site, the effect is to broaden the range of susceptible host cells.

If we consider all of the changes made to the spike protein in their entirety, then the probability that they should all have occurred by chance at the same time is vanishingly small. The odds would be reduced even further if we factored in several more observations which pertain to the other proteins of the virus.

To use an analogy: Wikipedia, that fount of unerring and benevolent knowledge, devotes an entire page to the “single-bullet theory” which has been advanced to support the official fairy tale about the murder of John F. Kennedy. Without batting an eye, Wikipedia explains:

*The single-bullet theory, also called magic-bullet theory by its critics, was introduced by the Warren Commission ... to explain what happened to the bullet that struck Kennedy in the back and exited through his throat. ... [Given that] Texas Governor John Connally was wounded and was seated ... in front of and slightly to the left of the president, the Commission concluded they were likely struck by the same bullet.*

The idea that all of the changes in the SARS-CoV-2 spike protein came about through natural evolution all at the same time has about the same degree of plausibility as the trajectory of this bullet. Belief in either of these absurd tales cannot be rationally justified.

## 1.4 SARS-CoV-2's purported ancestry (peer-reviewed science!)

- Several purported ancestral virus genomes show multiple hallmarks of having been faked in the lab
- Another supposed parental strain supposedly isolated from bats binds bat cell receptors very poorly
- Chinese researchers claim to have isolated several more related strains from Malaysian pangolins—but a separate study on several hundred pangolins has not found a single coronavirus in them

In their second study [5], Yan et al. examine—or rather, shred—the evidence which has been advanced to prop up the story of SARS-CoV-2's natural emergence. This evidence consists of a series of novel coronavirus genomes which exhibit a very high degree of sequence similarity to SARS-CoV-2. The closest relative among these is a strain named RaTG13 [12].

The RaTG13 virus strain is said to have been isolated from a sample of bat feces. If this were correct, then the nucleic acids obtained and amplified from this sample should contain a lot of bacterial DNA sequences, because stool consists largely of bacterial biomass. However, the available raw sequences contain only very small amounts of bacterial material. In addition, any animal DNA in these samples should mostly be derived from bat—but the sample contains DNA sequences from multiple other animal species.

The researchers who published the RaTG13 sequence maintain that the original sample has been used up entirely in the process of sequencing, and furthermore that the virus has not been grown in cell culture—in fact, that cultivation was not even *attempted*. This latter claim lacks any credibility—no virologist in the world would squander every last drop of a sample which contains a new virus without at least trying to grow it.

Another circumstance that proves the fraudulent nature of the RaTG13 strain is that the spike protein of this alleged bat virus *fails to bind the ACE2 receptors of horseshoe bats* [13]. The virus is said to have been obtained from a different bat species than the two species whose ACE2 receptors were used in this experiment. However, the receptors of all bat species should of course be very similar to each other. Thus, this result would imply that RaTG13 is not adapted to its own animal host, which is of course absurd and impossible.

## 1.5 Conclusion

- It is statistically impossible that all of the unique features in the SARS-CoV-2 genome arose naturally, that is, by chance
- The virus genome shows clear traces of the use of recombinant DNA techniques
- A fictitious natural lineage of the virus has been constructed in the mainstream scientific press that is based on more fraudulent science

To any clear-thinking person, the only possible conclusion from the findings presented in this chapter is that the evidence proves criminal intent beyond a reasonable doubt.

The reviewers who rejected Yan's publication made much of the fact that the ZC45 virus, which Yan suggests as the starting point ('backbone') for the construction of SARS-CoV-2, deviates from SARS-CoV-2 in about 10% of the genome [6]. Yan actually suggested that either ZC45 itself or an unpublished close relative of it was used as the starting point; indeed, the latter case seems much more likely. Beyond this single valid point, the criticisms raised by the referees are entirely lacking in substance.



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## Chapter 2

### COVID vaccines

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#### 2.1 Introduction

As of December 2020, the first COVID vaccine, an mRNA vaccine from Pfizer, has been rolled out in several countries, and others are nearing approval. The Federal Drug Administration (FDA) in the U.S. has granted an “emergency use authorization” (EAU) for this vaccine. The document on which this authorization is based is publicly available [14], and we will give it a good, hard look in the second half of this chapter. However, we will begin with some more general considerations.

##### 2.1.1 Goals of vaccination

- Protection of the vaccinee from severe disease (“relative immunity”) Example: attenuated tuberculosis live vaccine (no longer commonly used)
- Protection of the vaccinee from any infection (“sterilizing immunity”) Example: hepatitis B
- Protection of general population through “herd immunity” Examples: poliomyelitis, smallpox

Vaccines differ in their effectiveness. The minimum standard that we should demand is *relative immunity*—the vaccinee (vaccinated person) may still get infected with the pathogenic microbe in question, but the vaccination reduces the severity of the disease. Relative immunity is a quite common outcome of antibacterial vaccinations; the tuberculosis vaccine, which is an attenuated strain of the actual pathogenic bacterium (*Mycobacterium tuberculosis*), is a good example.

With diseases caused by viruses, we can often achieve *sterilizing immunity*. This means that the immune response induced by the vaccine prevents any and all propagation of the virus by the vaccinee. If sterilizing immunity prevails, the vaccine may be used to induce *herd immunity*—a sufficiently high degree of immunity in

the general population denies the wild-type pathogen in question the chance to reproduce effectively, and it may eventually disappear. Smallpox remains the only example of a virus that has been completely eradicated; this was achieved even though some individuals, for example those with neurodermatitis, had been exempt from vaccination.

If a vaccine cannot achieve sterilizing immunity, then it can't achieve herd immunity either; thus, there can be no justification for making such a vaccine mandatory.<sup>1</sup>

### 2.1.2 COVID-19 vaccination: executive summary

- Do we need vaccination for individual protection?
  - The pandemic is essentially over—the ongoing “second wave” is a hoax that is based on fraudulent test procedures
  - The infection with SARS-CoV-2 does not usually cause serious disease
  - Effective treatments exist for severe cases (but they were relentlessly maligned in the media and their use prohibited by officialdom)
- No COVID vaccine has yet been shown to induce sterilizing immunity—therefore, mandating vaccination “to protect others” is unjustifiable

It was always clear that the COVID pandemic would be over before an effective vaccine could be developed and properly tested. In vaccine development, the quick and easy part is always to cobble together some candidate vaccine that will induce some sort of immune response in a mouse; anyone with two years worth of training in molecular biology can do this. The hard part is to thoroughly establish the efficacy and safety of a vaccine in humans. This inevitably takes time—after all, a vaccination is not usually intended to provide only six months worth of immunity (although there are exceptions, e.g. with cholera vaccine given to those travelling to endemic areas). The shortcuts taken in case of the COVID vaccines mean that neither safety nor efficacy have been adequately established.

It is also striking to note the discrepancy between the attitude taken by officialdom towards several treatments for manifest cases that were found to be effective by many clinical practitioners. Scares were whipped up around drugs such as vitamins C and D, hydroxychloroquine, ivermectin, and budesonide, whose risks and other properties are well-understood from long-standing use against other diseases; and in some cases doctors were threatened with sanctions for continuing to use these treatments. In contrast, the vaccines, whose safety and efficacy are at best

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<sup>1</sup>If you are wondering where I stand on vaccinations in general: I believe that with several vaccines in current use the benefits outweigh the risks. This is the case for example with diphtheria, tetanus, hepatitis B, and poliomyelitis. The case for or against each vaccine must be made individually; both a sweeping “yes” and a sweeping “no” are wrong. A separate question is whether commercial vaccine preparations are always as safe and benign as they could be; without going into detail, I will just say that I see room for improvement, particularly in the selection of adjuvants.

unknown, have been presented to the public as mankind’s only hope for overcoming COVID and returning to a normal life.

In a sane world, none of this would have happened—the danger of COVID would not have been blown out of proportion, and nobody would even have started on developing a vaccine, never mind filing for emergency use authorization. The remainder of this chapter should accordingly be irrelevant. Sadly, the world is not a sane place right now.

## 2.2 Antiviral vaccination methods

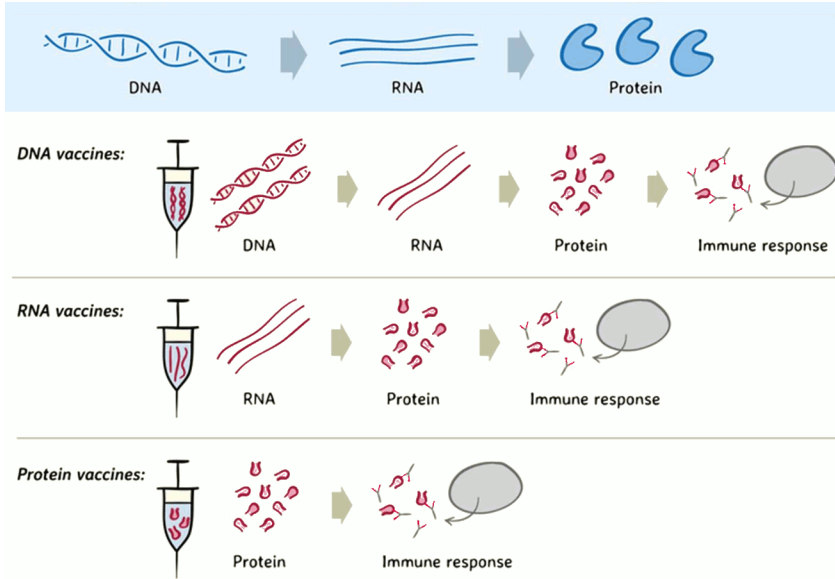
	Method	Example	Risks/drawbacks	Benefits
Conventional	Attenuated live virus	Poliomyelitis (Sabin)	Genetic instability	Lasting immunity
	Chemically inactivated virus	Poliomyelitis (Salk)	Multiple injections	Not infectious
	Recombinant viral proteins	Hepatitis B	Multiple injections	Not infectious
Non-conventional	mRNA (lipid nanoparticles)	COVID-19 Pfizer, Moderna	Immune pathology	Promotion of cellular immunity
	DNA (viral vector)	COVID-19 AstraZeneca	Immune pathology; genetic modification	Promotion of cellular immunity

All of the conventional types of vaccines listed in this table are currently widely used in practical medicine, and their advantages, limitations, and risks are quite well understood. In contrast, none of the non-conventional vaccine types, which are based on purified nucleic acids (DNA or mRNA) or on viral vectors, have so far passed a regular approval process for clinical use. The recent approval of Pfizer’s COVID vaccine was granted only under the rather loose rules of “emergency use authorization.”

In practical research and development, most people will follow the common sense rule to change only one parameter or aspect at a time. In the context of vaccine development, this would mean to try either an old vaccination method on a new virus, or a new vaccine paradigm on an old virus, whose pathogenesis you already thoroughly understand, and for which established vaccines exist that can be used as a benchmark for testing the new vaccine. The push for solving the imaginary and contrived COVID problem with new vaccination methods, and to do so in record time, does not pass the smell test. Those who are doing the pushing either are aware that the problem is indeed fictitious, or they do not care whether or not the problem really is solved. We will see below that the non-conventional DNA and RNA vaccines

against COVID that are currently being foisted upon mankind do not measure up to conventional ones used against other viruses.

### 2.2.1 The “central dogma of molecular biology” and its application to vaccines



This slide (adapted from [15]) illustrates the principles behind the use of DNA, RNA, and recombinant viral protein vaccines. In any living cell, genetic information is stored in the form of double-stranded DNA. The DNA is transcribed (copied) into single-stranded messenger RNA (mRNA), which is then translated into proteins.

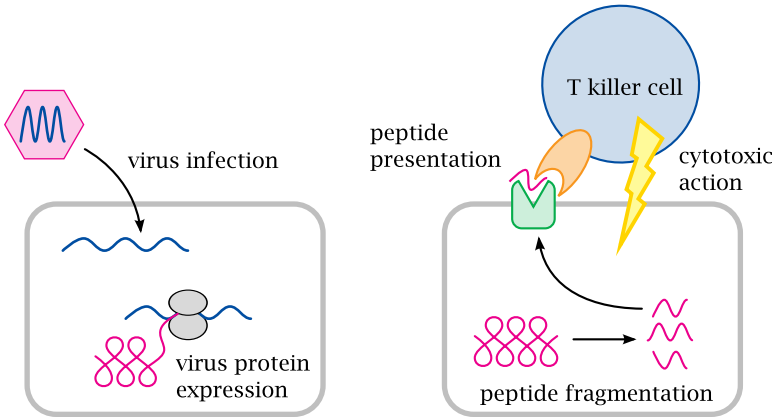
DNA vaccines contain the genes which encode the viral proteins against which we want to induce an immune response; they rely on the cellular machinery for both transcription to mRNA and translation to protein. Such DNA molecules *might* insert themselves into the chromosomal DNA of the host cell.

Messenger RNA vaccines rely on the cell only for the protein translation step. Since no alien DNA is introduced, the risk of altering the chromosomal DNA is small, although it cannot be absolutely ruled out. In contrast, this risk is non-existent with the use of recombinant proteins; there is no known mechanism for translating a protein backwards into RNA and then DNA.

### 2.2.2 Outline of T lymphocyte function in antiviral immune responses

From the foregoing, it is apparent that recombinant protein vaccines are the safe choice. Why, then, would one entertain the use of DNA or mRNA instead? The rationale is that nucleic acids better mimic a viral infection—much like virus-infected cells, a cell that has taken up a nucleic acid vaccine will synthesize the viral proteins within. Some of the viral protein molecules will be broken down again. The resulting small fragments (peptides) are exposed on the cell surface, in conjunction with a specific “anchor” molecule (a class I HLA molecule; shown in green in this illustra-

tion). The complex of viral peptide and the HLA molecule is recognized by a T killer lymphocyte which happens to possess a matching T cell receptor (shown in orange). This recognition triggers the T cell to destroy the virus-infected cell. Moreover, it also causes the T cell to divide and grow in numbers. This activation and expansion of the T cells takes from one to two weeks; once the T cells are out in force, they usually manage to snuff out the virus infection within a few days. This final battle between the virus infection and the immune system is usually accompanied by fever and other symptoms of inflammation; the lag time of the immune response corresponds to the incubation period.



As we will see, however, this potential advantage of the nucleic acid vaccines also gives rise to increased risk of adverse reactions.

### 2.2.3 Why are nucleic acid (DNA and RNA) vaccines more likely to cause immune pathology?

- An antiviral immune response has two elements:
  - T killer cells attack and destroy virus-infected cells
  - Antibodies bind and block viral surface proteins
- If an immune person is reinfected with the real virus or exposed to a conventional vaccine, these will be intercepted by antibodies—live viruses will be prevented from entering cells and multiplying
- Nucleic acids which are unaccompanied by the viral proteins will enter the cells regardless of antibodies
- The cells will produce the viral proteins encoded by those nucleic acids and then be attacked by the T-lymphocytes

In any viral infection, the body produces not only cytotoxic T-lymphocytes (T-killer cells), but also antibodies. These are extracellular proteins, which will swiftly bind to the surface of the virus particles and thereby prevent them from entering cells

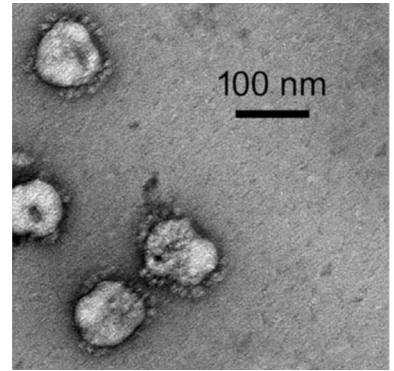


and then multiplying within them. While the removal of these inactive extracellular antibody-virus complexes may be accompanied by some inflammatory symptoms, there usually is no severe harm.

In contrast, if an immune person is injected with a nucleic acid (DNA or RNA) vaccine, these nucleic acids will not be recognized and neutralized by the antibodies. Thus, they will be permitted to enter the cells, causing the latter to produce viral proteins and then to be attacked and destroyed by the T-killer cells. This could happen in any organ, but the most severe consequences of the destruction of a limited number of cells must be expected with the central nervous system. Moreover, in patients who have very recently been infected with the wild-type virus and therefore have a very active immune response going, there might be severe acute symptoms of inflammation.

### 2.3 An inactivated virus vaccine from China

- SARS-CoV-2 isolated from human patients, grown in cell culture, chemically inactivated
- Tested in several animal models, most notably rhesus monkeys
- Animals were vaccinated, challenged with live virus, and sacrificed
- Virus load in throat and anal swabs reduced (but not to zero)
- Pneumonia was mitigated but not entirely prevented



This slide summarizes the results of an animal study reported by Wang et al. [16]. The title of the study claims “potent protection” of the animals against SARS-CoV-2, which is not borne out by the actual results. However, the effectiveness of this vaccine is as good as or better than that of the nucleic acid vaccines that we will consider below. Thus, if there were a shred of common sense and regard for our health left among those who are promoting COVID vaccination, this is the kind of vaccine they should be advocating. The big push for the nucleic acid vaccines makes it plain that another agenda is at play.

The electron micrograph in the slide shows quite credible coronavirus particles. Thus, unless this study is altogether fraudulent, it does provide solid proof of the existence of the SARS-CoV-2 virus.

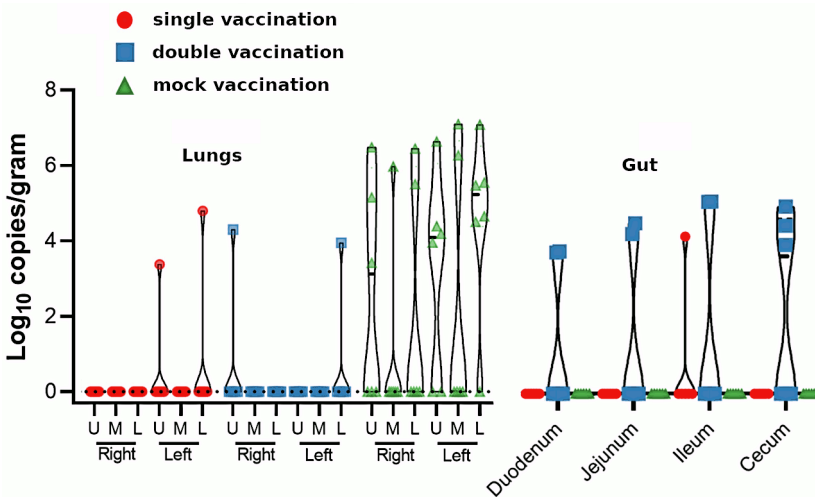
### 2.4 AstraZeneca: the vaccine from hell

- Vaccine design: gene encoding SARS-CoV-2 spike protein inserted into non-replicating “vector” virus
- Tepid results in animal experiments

- partial protection of rhesus monkeys from pneumonia
- very weak antibody response
- no reduction of virus load in nasal swabs

The failure of this vaccine to reduce viral replication in the mucous membranes of the nose means that it does absolutely nothing to “stop the spread of COVID”—getting vaccinated *might* reduce disease severity in some people, but these same people will still catch and further disseminate the infection. Thus, there can be absolutely no justification for making this vaccine mandatory. Nevertheless, the EU has already pre-ordered about 400 million doses for its citizens [17], which strongly suggests that mandatory vaccination is in the works—even though this vaccine has not even received emergency use authorization.

#### 2.4.1 The AstraZeneca vaccine inhibits viral replication in the lungs but induces replication in the gut



This slide (adapted from [18]) illustrates a surprising finding from animal studies—double vaccination causes replication of the virus in the gastrointestinal tract, which is uniformly absent in mock-vaccinated animals.<sup>2</sup>

While the transmission to other people through the fecal route seems unlikely, this finding is nevertheless significant: it is entirely unexpected, and the study does not provide any explanation. This illustrates that the researchers do not really understand what they have been doing, and it reinforces the earlier point that it is not a good idea to tackle a new virus with unproven technology, particularly when time is perceived as being of the essence.

<sup>2</sup>The  $y$  axis indicates the logarithm of the number of genome copies detected in various tissues. The letters U, M, and L represent the upper, middle, and lower lobes of the left and right lungs of the animals. Duodenum, jejunum, and ileum are segments of the small intestine, whereas the cecum is part of the large intestine.

In saner times, and with manufacturer liability, a vaccine with such puzzling and overall disappointing results would never even have entered human trials. Now, however, this vaccine has been pushed through clinical trials on a recklessly accelerated schedule. Even these shortened trials, however, have turned up some worrisome results.

### 2.4.2 The AstraZeneca vaccine: clinical trials

- Intense flu-like reactions (fever above 38, sometimes 39°C)
- Two cases of transverse myelitis in early clinical trials
- Clinical trials were paused, but resumed after one case of transverse myelitis had been retroactively reclassified as “multiple sclerosis”

While fever as such is transient and tractable, this finding indicates a rather intense inflammatory reaction. One can therefore expect more severe manifestations in some patients, and the two cases of transverse myelitis confirm this expectation. The intensity of the inflammatory reactions forms a striking contrast to the very limited degree of relative immunity conferred by the vaccine; by normal standards, this vaccine is an outright and complete failure.

The retrospective change of the diagnosis of the first presumptive case from transverse myelitis to multiple sclerosis (MS) is suspicious. If that patient had had a pre-existing MS diagnosis, then he or she would most likely have been excluded from the study to begin with; and if not, the clinical manifestation would have been chalked up to the MS right away and not only after the second case of transverse myelitis had occurred. The people behind this study have repeatedly been criticized for their lack of transparency [19, 20] and are altogether untrustworthy.

### 2.4.3 Some background on transverse myelitis

- NIH estimates 1,400 annual cases in United states (roughly one case in 200,000 people)
- Symptoms: paraparesis/paraplegia
- Causes:
  - various viral infections
  - autoimmune disease, including vaccine reactions
- Prognosis: some patients recover fully, others partially, yet others not at all

Transverse myelitis is an inflammatory disorder that affects the spinal cord and functionally severs it, causing paraplegia or, if the severance is incomplete, paraparesis. Recovery is slow, and some patients recover only partially or not at all.

The disease is rare in the general population, but a connection to autoimmune disease, sometimes induced by vaccination, is known. The occurrence of two such

cases among the unknown number of test persons in the AstraZeneca study is very worrisome. If the manufacturer were liable for such cases, management would not dream about calling this toxic waste a “vaccine” and unleashing it on mankind.

## 2.5 The Pfizer vaccine: lies, damn lies, and statistics

- First vaccine to U.S. market via “emergency use approval” (FDA)
- Vaccine design: mRNA encoding the receptor-binding domain of the viral spike protein
- Combined with lipid mixture that facilitates cellular uptake
- Two intramuscular injections, 3-6 weeks apart
- Tested in rushed phase 3 trials that involved some 40,000 persons overall; one half received vaccine, the other half received placebo
- Vaccine approved in Canada as well, but apparently no substantial documentation available from Health Canada
- FDA documentation full of holes, contradictions, gimmicks, and outright lies

We now turn to the vaccine produced by Pfizer, which was recently approved for “emergency use” in the United States and several other countries, including Canada. While the FDA released a 53 page memo to support its decision [14], I have not found any comparable document from Health Canada, and therefore will focus here on the data and the claims contained in the FDA document.

The vaccine contains a synthetic messenger RNA, which encodes the receptor-binding domain of the viral spike protein; this is the part that mediates the attachment to host cell receptors (see slide 1.2.8), and its blockade by antibodies may therefore be expected to protect from the infection. In contrast, such a small part of the virus particle is not likely to induce very strong cell-mediated immunity (T-killer cells). Therefore, the choice of an mRNA vaccine rather than a protein vaccine is puzzling; if the goal is to induce neutralizing antibodies, protein vaccines work perfectly well, as is illustrated by the tried and true vaccines against tetanus toxin and diphtheria toxin.

### 2.5.1 “The vaccine is 95% effective”—sure sounds great, but what does it mean?

The observation of the study participants was rather limited: they were asked to visit the doctor’s office if they experienced symptoms of respiratory illness, whereupon nasal swabs would be taken and tested for SARS-CoV-2 by PCR. If that test produced a positive result, and if the patient indeed exhibited one or more generic symptoms of flu-like disease, then a case of COVID was diagnosed. Unsurprisingly, no specifics about the PCR protocols used are detailed; only a number of commercial suppliers of test kits are named.

The claim of a “95% efficacy” is based on the observation that, one week after the second injection or later, 8 cases of COVID occurred in vaccinated group, whereas

162 such cases reportedly occurred in the placebo group. If we take those 162 cases as 100%, then the 8 cases in the vaccinated group correspond to 5%; the difference of 95% is passed off as the vaccine’s efficacy.

	Vaccine		Placebo		Ratio
	Cases	%	Cases	%	
Visit to doctor, followed by positive PCR	8	0.044	162	0.89	0.05

- 0.85% of vaccinated patients are saved from having a positive COVID PCR test
- Average observation time: 7.6 weeks (study emphasizes median of >2 months)
- No data on number of visits to doctor’s office overall
- No data on number of PCR tests performed in each group—thus, we can’t judge the effect of false positives

What the FDA memo does not tell us is the number of visits to the doctor that occurred in each group, and the number of PCR tests performed on the patients in question. However, considering the high incidence of flu-like symptoms in the vaccinated group, it is not unlikely that the number of transiently sick individuals was greater among the vaccinated than among the placebo group. We might tentatively accept that the vaccine saved 0.85% of the recipients from the traumatic experience of a positive COVID PCR test—however, as we will see below, this claim rests on dubious data.

The study also does not provide any information on the durability of the claimed immunity. It can’t, since these “phase 3 clinical trials” were a rushed affair: the average time of observation after the second of two vaccine shots was only 7.6 weeks (but the study talks only about the median time of observation, claiming that it exceeded 2 months). Also lacking are observational data on long-term safety. While it is true that most adverse reactions typically occur within a few weeks of the vaccination, the complete lack of long-term follow-up before the onset of mass vaccination is worrisome.

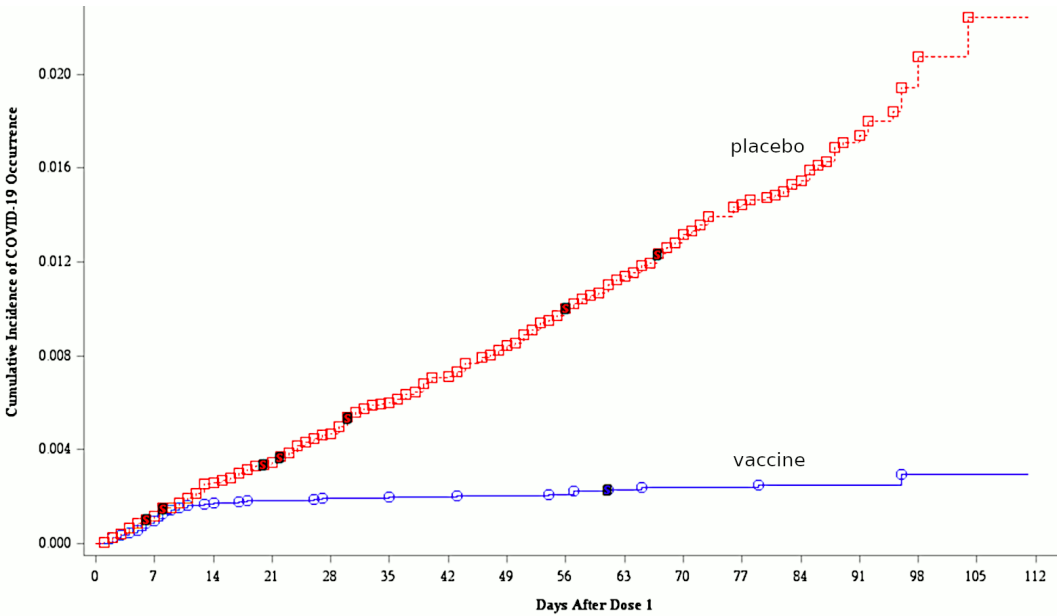
**2.5.2 COVID-19 incidence over time in vaccinated and placebo groups**

We will now take a closer look at the data contained in the FDA document. The plot shown here is from Figure 2 of the document. The *x* axis indicates the days after the first injection; the second injection took place between days 19 and 42 (vaccinees who received the second shot outside this time interval were excluded from the evaluation). The *y* axis shows the cumulative incidence of “COVID,” that is, some non-specific symptom or other and a positive PCR test.

In the placebo group, the cumulative incidence trends up pretty much in a straight line, which means that the average number of cases per day or per week remained constant and was not affected by the mock injections. In the injection

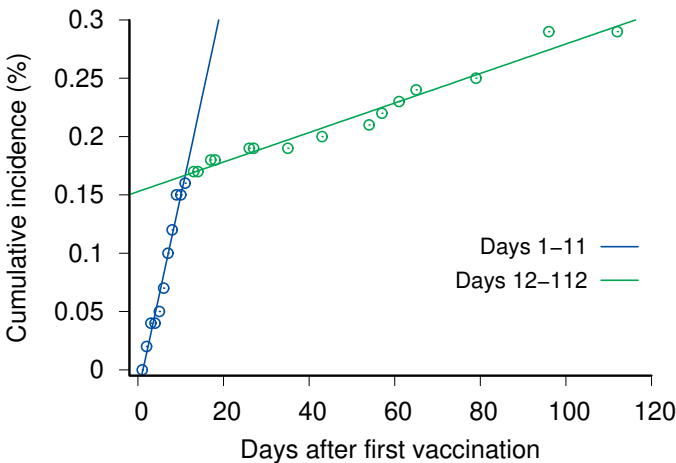


group, we see a closely similar trend for the first 11 days after the first injection; however, beginning with the twelfth day, the rate of new cases drops abruptly. This is highlighted in the subsequent slide (2.5.3).



### 2.5.3 Sudden onset of immunity on day 12 after first injection

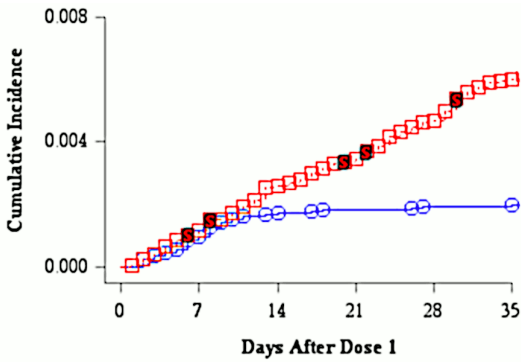
This slide magnifies the vaccine curve from the preceding slide to show that the change in the incidence is indeed abrupt—we have a high incidence up to day 11, and a much lower one beginning with day 12. Each stage is well approximated by a linear regression graph.<sup>3</sup>



<sup>3</sup>According to the vaccine's claimed efficacy of 95%, the ratio of the two slopes should be 20, but it is only 13.3; this corresponds to an efficacy of 92.5%.

The abrupt change in incidence would imply an equally sudden onset of immunity. Moreover, this sudden onset occurs one week or more *before* the second injection. This is not biologically plausible—onset of immunity would be more gradual in real life, and moreover maximal immunity is usually attained only after two or more injections. Thus, these data must have been manipulated or wholly fabricated.

2.5.4 The FDA memo contradicts itself on COVID incidence after vaccination



	vaccine	placebo
Negative on day 0	93.1%	93.0%
Negative on day 35	85.6%	85.0%
Conversion	7.5%	8%
Excluded for “other protocol deviations”	1.4%	0.3%

Aside from the graphical representation of COVID “cases” in the vaccine and placebo groups, the FDA memo also reports in its Table 2 the percentages of persons without evidence of prior COVID infection on the day of the first injection, as well as two weeks after the second one. The regular date for the second injection was 21 days after the first, although persons who were received it between days 19 and 42 were included in the evaluation. Assuming that most persons were injected on or near day 21, we can calculate how many people had turned positive between days 0 and 35.<sup>4</sup> These numbers turn out to be almost identical in both groups and are incompatible with the graph. This reinforces the conclusion that the claimed efficacy data are fraudulent.

Furthermore, on page 28, the FDA study states that “only 3% of participants had evidence of prior infection at study enrollment.” This again contradicts the numbers quoted here. This is more evidence of fabrication and falsification. That the FDA should have overlooked such glaring discrepancies proves that the entire review and approval process was a farce.

<sup>4</sup>A “positive” baseline status is explicated as follows (see page 27): “Positive N-binding antibody result at Visit 1, positive NAAT result at Visit 1, or medical history of COVID-19.” The “N-binding” antibody is one that binds to the complex of the viral RNA and its associated protein (the “N + gRNA” element in the illustration in slide 1.2.1). The acronym “NAAT” means “nucleic acid amplification test,” which is the same as PCR, which is now generally known to be unreliable. The “medical history of COVID” has its own problems, of course, since COVID-19 has no unique, characteristic symptoms—a number of respiratory viruses can cause the same clinical picture.

### 2.5.5 Hold the presses! The Pfizer vaccine protects from COVID more effectively than does prior infection with the virus!

	Vaccine			Placebo		
	Total	Cases	Incidence (%)	Total	Cases	Incidence (%)
All subjects	19965	9		20172	169	
Initially negative	18198	<b>8</b>	<b>0.044</b>	18325	162	
Previously infected	1767	1		<b>1847</b>	<b>7</b>	<b>0.38</b>

While blatantly fabricating the evidence of protection pertaining to those without evidence of previous COVID infection is bad enough, Pfizer does manage to top this achievement by also claiming that their vaccine protects from an infection with the wild type virus more reliably than a prior infection with the wild type virus itself. This can be inferred from the difference between the protection achieved in all subjects (Table 6 of the report) and those without previous infection (Table 7).

Ask yourself: does this happen with measles? mumps? smallpox? rubella? Of course, it does not—and neither will it be the case with this man-made virus. The only conclusion to draw from these numbers is that the liars in the employ of Pfizer are particularly inept, and the FDA’s “reviewers” were asleep at the wheel.

### 2.5.6 Pfizer vaccine: adverse reactions

- no excess total mortality in vaccine group: 2 deaths in vaccine group, 4 in placebo group; several deaths seem unrelated (heart attack, stroke)
- “In the vaccine group, one participant with baseline obesity and pre-existing atherosclerosis died 3 days after Dose 1”
- four cases of Bell’s (one-sided facial) palsy in vaccine group, zero in placebo (expected: 0.32)—study claims that “the four cases in the vaccine group do not represent a frequency above that expected in the general population”
- plenty of patients with fever, headaches, muscle and joint pain etc.—flu-like disease more common in vaccine than placebo group
- No distinction is made between initially negative and previously infected vaccinees

Among the four fatalities in the placebo group, two were due to stroke and heart attack, respectively, whereas two are ascribed to unknown causes. While we can of course assume that they were indeed unrelated to the vaccine, it is odd that no complete records were compiled as to the exact cause of these deaths; after all, while the study was ongoing, one should have considered that similar cases might accrue in the vaccinee group as well during the remaining study period.

In the vaccine group, one participant died 62 days after the second vaccination due to “cardiac arrest,” which we can accept as likely unrelated to the vaccine. However, the second patient is said to have died “from arteriosclerosis” 3 days after the first vaccination. This information is woefully inadequate. One does not die from arteriosclerosis; instead, one does die from acute events caused by arteriosclerosis, such as a heart attack or a stroke. Also, the short time interval between the vaccination and the death raises the question what exactly went down—did the patient in question suffer an immediate reaction to the injection, to which he succumbed 3 days later, or was he initially fine and then suddenly fell ill and died at home? Again, this kind of “documentation” is wholly inadequate and should have been rejected outright.

Regarding Bell’s palsy, the expected incidence of 0.32 in each group can easily be calculated from information provided for free by the NIH [21] and the average post-vaccination observation period of 7.6 weeks. The claim that the observed number of 4 cases is “within expectation” is another blatant lie.

A glaring omission of the Pfizer study, and of the AstraZeneca study as well, is that adverse reactions are not tabulated separately for patients with and without prior infection with the wild type virus. Allergic and inflammatory reactions can be expected to be more severe in those with prior infection. At the same time, these patients are very unlikely to benefit from the vaccination—just like those of us who experienced measles as children will not derive any benefit from being vaccinated against it. If the purpose of this vaccination campaign were indeed to safeguard the health of the population, we would be advised to get vaccinated only after testing negative for COVID. That this advice is not given shows once more that another agenda is at play.

### 2.5.7 Pfizer vaccine: summary

- Claimed reduction in COVID incidence is fabricated
- Adverse reactions are reported incompletely and dishonestly
- “A larger number of individuals at high risk of COVID-19 and higher attack rates would be needed to confirm efficacy of the vaccine against mortality.”

The quote in the last item says that there really is no emergency, and thus no justification for emergency use. The application for emergency use should never have been granted—that it was granted shows that the FDA approval process is corrupt and has broken down.

The lack of attention to the potential harm of vaccinating those who were already infected with the wild type virus rounds out the picture—this vaccine is not fit for medical use, and it is not meant to be. Like everything else connected with this plandemic, it is as phony as a three dollar bill—or, rather, make that a 30 billion dollar bill.

2.5.8 Adverse reactions to the Pfizer vaccine: initial results

### V-safe Active Surveillance for COVID-19 Vaccines

	Dec 14	Dec 15	Dec 16	Dec 17	Dec 18*
Registrants with recorded 1 <sup>st</sup> dose	679	6,090	27,823	67,963	112,807
Health Impact Events**	3	50	373	1,476	3,150
Pregnancies at time of vaccination	5	29	103	286	514

\*Dec 18, 5:30 pm EST

\*\*unable to perform normal daily activities, unable to work, required care from doctor or health care professional

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This slide summarizes the frequency of adverse reactions during the first few days of application in the United States. According to this information, which is sourced from the CDC [22], approximately 3% of all vaccinees experienced reactions severe enough to require medical assistance and prevent them from working or other normal daily activities. This outcome confirms unambiguously that this vaccine is not fit for medical use. The use of this vaccine must be halted immediately.

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