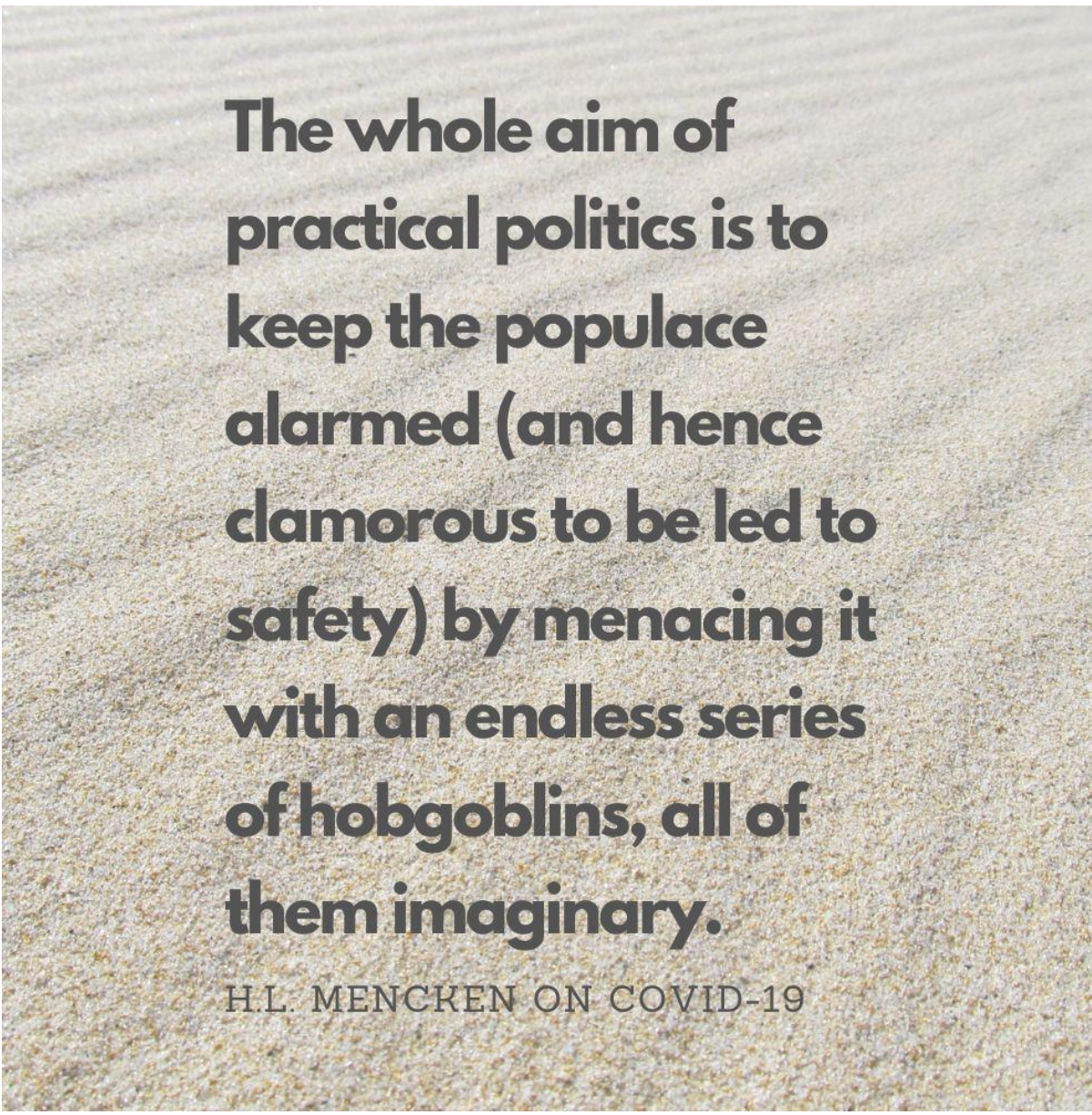
Notes on COVID



Michael Palmer, MD

Preface

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

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.

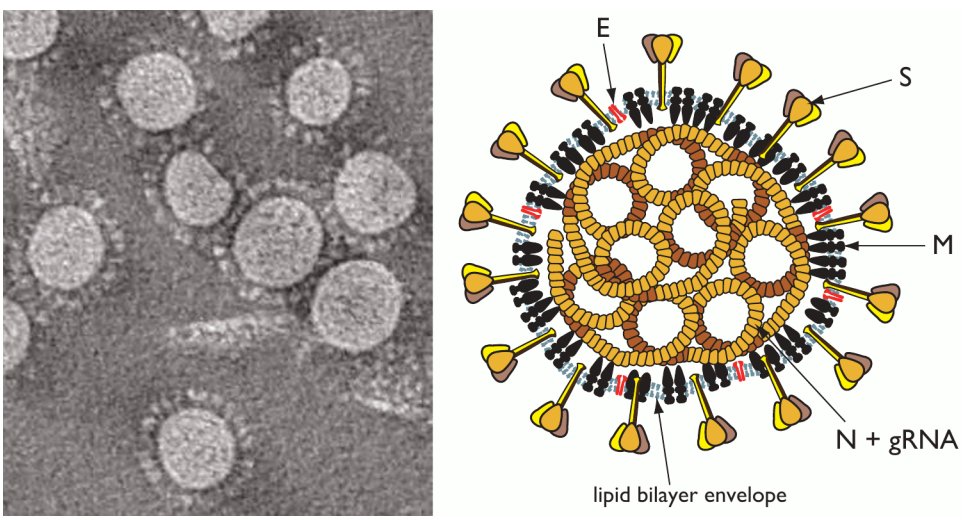
- MD/PhD virologist from Hong Kong University—recently fled to U.S., apparently now under Steve Bannon's protection
- I suspect that her back story contains some inaccuracies, but that is unrelated to scientific substance of her work
- Her papers have not been published by peer-reviewed journals—but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

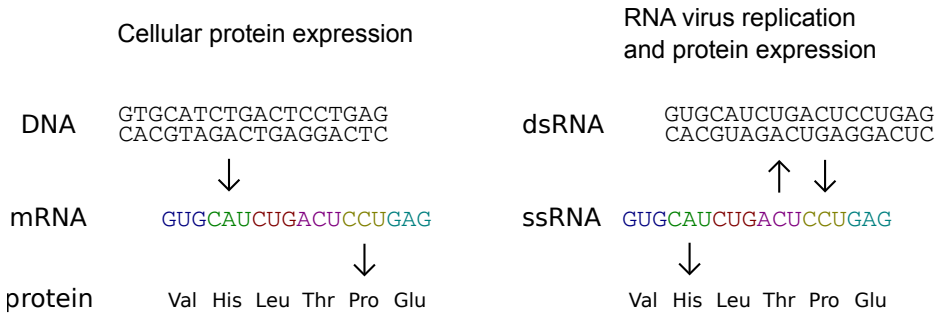
1.2.1 Structure of coronavirus particles



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).

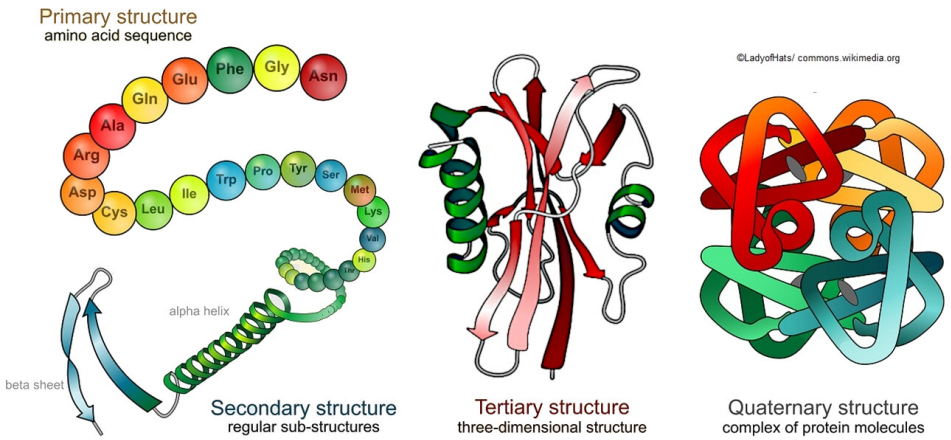
We will note here that it is possible in vitro to use PCR (polymerase chain reaction) to create a double-stranded DNA copy of a single-stranded RNA virus genome. From such a copy, the cellular RNA polymerase, whose regular job it is to transcribe cellular DNA into mRNA, can make single-stranded RNA copies 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.

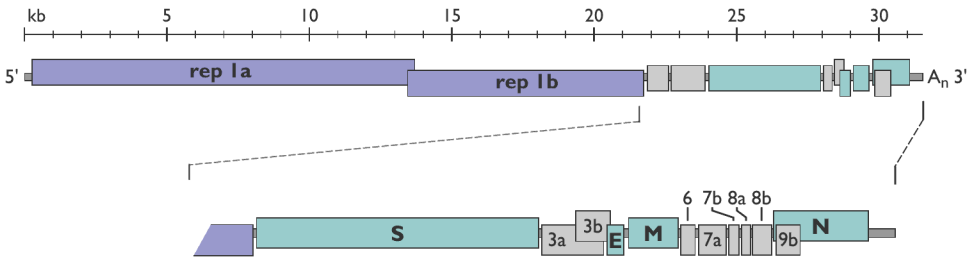
Within each protein molecule, all amino acids are connected like pearls on a string. However, this string will soon fold back upon itself, driven by mutual

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

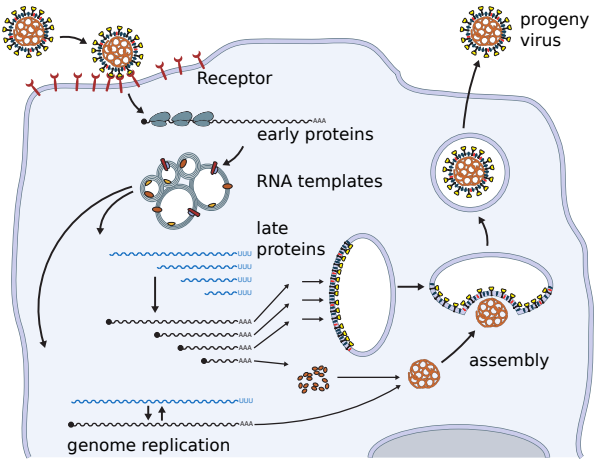


This map, which pertains to the SARS virus, is only meant to illustrate that the single RNA molecule contains the genes for all proteins that are needed to replicate the RNA (purple), to form the virus particle (green), and for several auxiliary functions (gray).

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

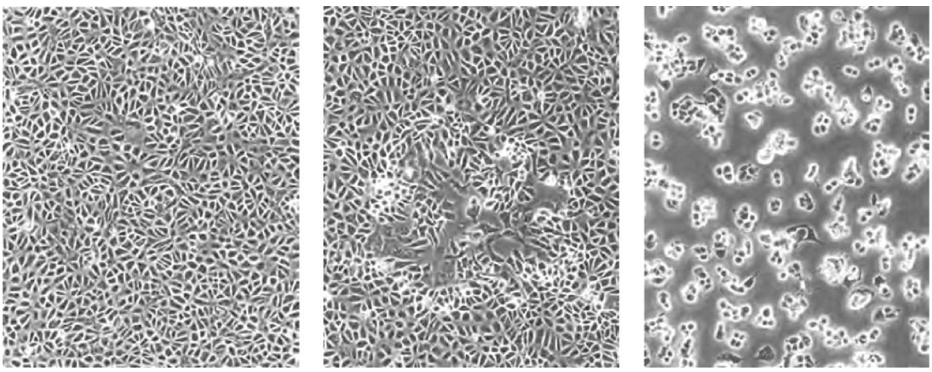
- virus particle binds to cellular receptors
- entry and uncoating of the RNA genome
- synthesis of early proteins for use in subsequent stages
- synthesis of late proteins and of RNA copies
- assembly of structural proteins and RNA genome into progeny virus
- exit of progeny virus



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.

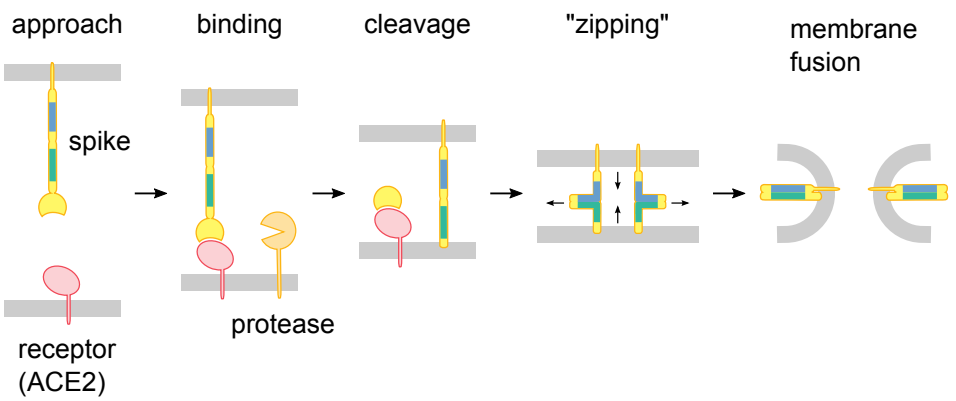
1.2.6 Virus replication in cell culture: the cytopathic effect



1.3 The structure and function of the spike protein

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

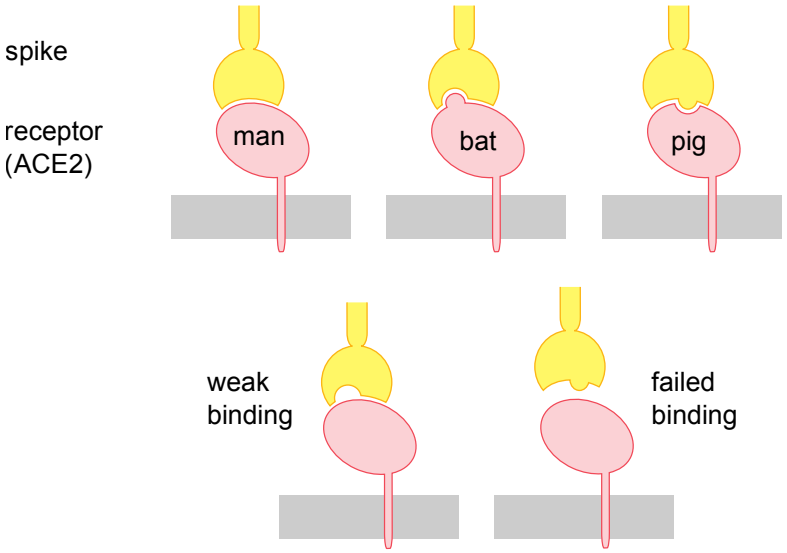
We had noted that the spike protein mediates not only binding but also the fusion of the viral envelope to the cell membrane. This second step is triggered when a host cell *protease*, that is, a protein which cleaves other proteins, does just this to the spike protein. The truncated spike protein then '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.



1.3.2 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.

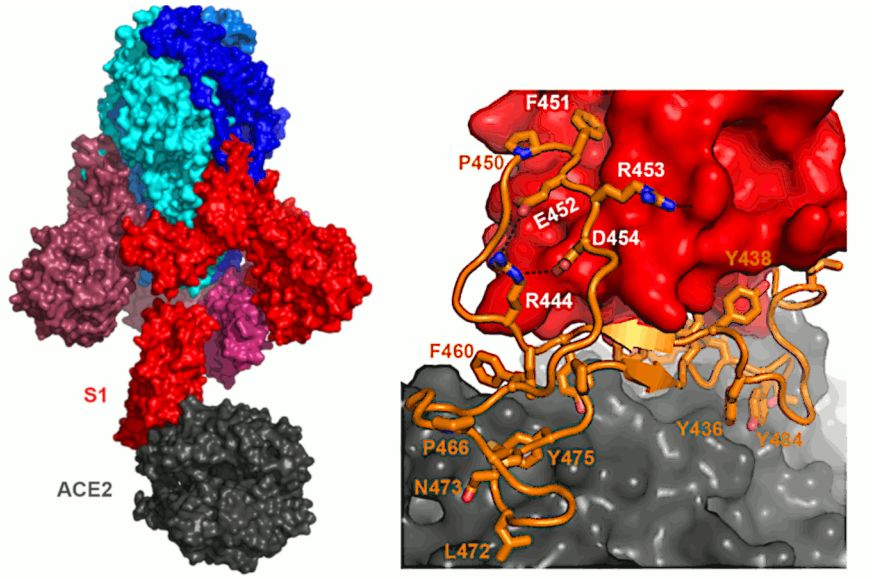


A rapid evolution towards improved receptor binding was indeed observed with the original SARS virus in the early 2000s. In contrast, with SARS-CoV-2, the affinity for the human receptor was already very high when the first strains were isolated for human patients. That means one of two things—either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

1.3.3 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.

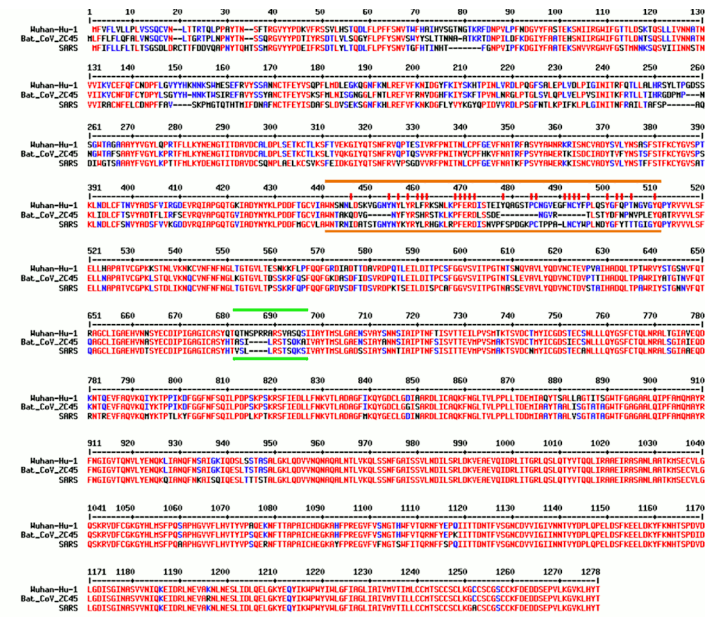
Below, we will refer to this part of the spike protein as the *receptor-binding domain* (RBD).



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

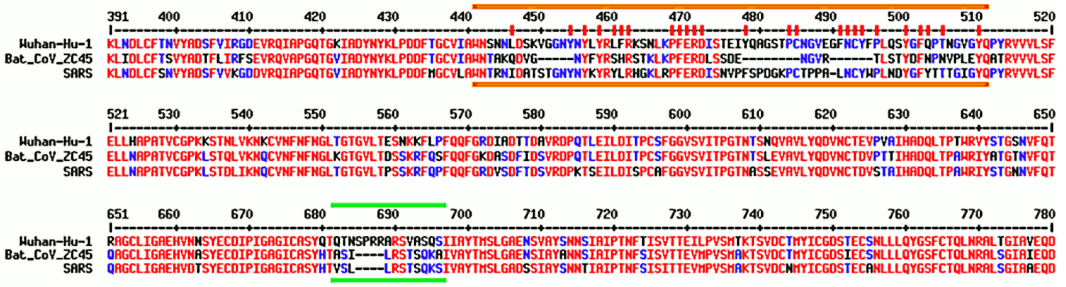
This slide (modified from [4]) aligns the sequences of three coronavirus strains. They include a SARS-CoV-2 strain from Wuhan, a related virus (ZC45) which Yan et al. [4] name as the most likely starting point for the creation of SARS-CoV-2, and the original SARS virus. In earlier slides, each amino 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 the beginning of the sequence than near its end. In their analysis, Yan et al. [4] focus on the two variable stretches highlighted with orange and green bars, respectively.



1.3.5 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 1.3.3. 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.



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

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 1.3.1). 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.

```
Human SARS-CoV BJ01          655 - GICASYHTVSLL----RSTS - 670
Human SARS-CoV CUHK-W1       655 - GICASYHTVSLL----RSTS - 670
Human SARS-CoV Tor2          655 - GICASYHTVSLL----RSTS - 670
Human SARS-CoV Frankfurt-1   655 - GICASYHTVSLL----RSTS - 670
Human SARS-CoV Urbani        655 - GICASYHTVSLL----RSTS - 670
Civet SARS-CoV civet020      655 - GICASYHTVSSL----RSTS - 670
Civet SARS-CoV sz16          655 - GICASYHTVSSL----RSTS - 670
Racoon dog SARS-CoV A030     655 - GICASYHTVSSL----RSTS - 670
SARS-CoV-2                   669 - GICASYQTQTNSPRRARSVA - 688
Pangolin CoV MP789           n/a - GICASYQTQTNS----RSVS - n/a
Bat SARSr-CoV RaTG13         669 - GICASYQTQTNS----RSVA - 684
Bat SARSr-CoV LYRa11         659 - GICASYHTASLL----RNTG - 674
Bat SARSr-CoV LYRa3          659 - GICASYHTASLL----RNTG - 674
Bat SARSr-CoV RsSHC014       656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs4084         656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV WIV1           656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs3367         656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs7327         656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs9401         656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs4231         655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV WIV16          655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV Rs4874         655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV ZC45           646 - GICASYHTASIL----RSTS - 661
Bat SARSr-CoV ZXC21          645 - GICASYHTASIL----RSTG - 660
Bat SARSr-CoV Rf4092         634 - GICASYHTASTL----RGVG - 649
Bat SARSr-CoV Rf/JL2012      636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV JTMC15         636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV 16BO133        636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV B15-21         636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV YN2013         633 - GICASYHTASTL----RSIG - 648
Bat SARSr-CoV Anlong-103     633 - GICASYHTASTL----RSVG - 648
Bat SARSr-CoV Rp/Shaanxi2011 640 - GICASYHTASVL----RSTG - 655
Bat SARSr-CoV Rs/HuB2013     641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV YNLF/34C       641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV YNLF/31C       641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV Rf1            641 - GICASYHTASHL----RSTG - 656
Bat SARSr-CoV 273            641 - GICASYHTASHL----RSTG - 656
Bat SARSr-CoV Rf/SX2013      639 - GICASYHTASLL----RSTG - 654
Bat SARSr-CoV Rf/HeB2013     641 - GICASYHTASLL----RSTG - 656
Bat SARSr-CoV Cp/Yunnan2011  641 - GICASYHTASLL----RNTG - 656
Bat SARSr-CoV Rs672          641 - GICASYHTASTL----RSVG - 656
Bat SARSr-CoV Rs4255         641 - GICASYHTASTL----RSVG - 656
Bat SARSr-CoV 4081           641 - GICASYHTASTL----RSVG - 656
Bat SARSr-CoV Rm1            641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV 279            641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV Rs/GX2013      642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Rs806          641 - GICASYHTASLL----RSTG - 656
Bat SARSr-CoV HKU3-1         642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Longquan-140   642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Rp3            641 - GICASYHTASTL----RSVG - 656
Bat SARSr-CoV Rs4247         642 - GICASYHTASTL----RSVG - 657
Bat SARSr-CoV Rs4237         641 - GICASYHTASTL----RSVG - 656
Bat SARSr-CoV As6526         641 - GICASYHTASTL----RSVG - 656
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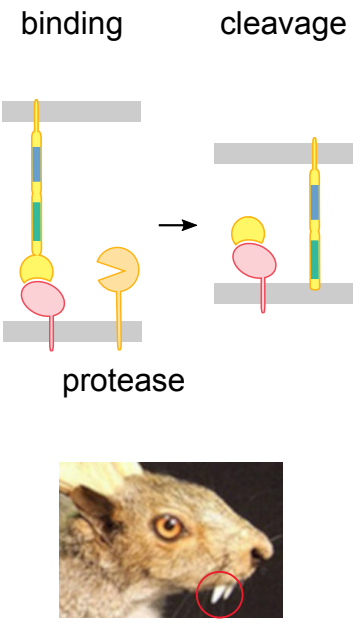
SARS-COV 2



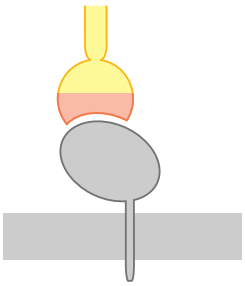
While furin cleavage sites similar to that of the SARS-CoV-2 spike protein do occur in some distantly related coronavirus strains [7], they do not occur among the SARS family (see sequence alignment on the left) or other virus strains closely related to SARS-CoV-2. The absence of a furin site in those 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*,[1] 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.[2] Such genetic exchange will occur efficiently only between strains which 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] No connection to Anthony Fauci.

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

1.3.7 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.

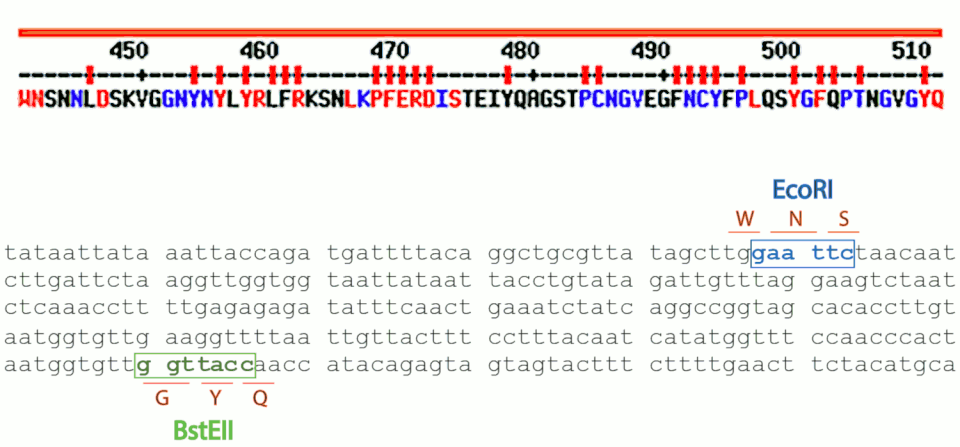
The red markers in the line above the three sequences indicate amino acid positions that are understood to be crucial for the interaction of the spike protein with the human ACE2 receptor. If we focus on SARS and SARS-CoV-2 only, we see that most of the positions flagged by red markers are occupied by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

1.3.8 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.[3]

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.

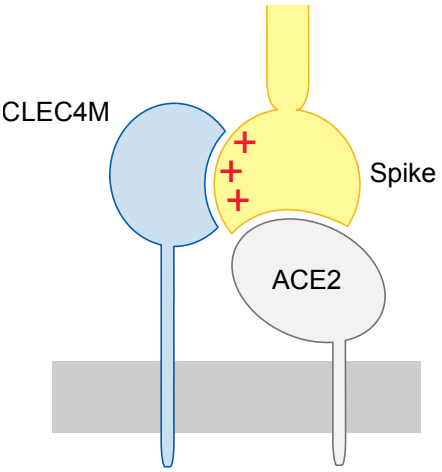
Lauring's estimate of such a site occurring once every 4096 bases assumes that all 4 bases (building blocks) occur with equal frequency, which is incorrect; but his estimated probability is not too far off the actual value. Much more gravely, however, Lauring ignores that *two* such sites occur here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

1.3.9 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

[3] 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.

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.

Chapter 2

COVID vaccines

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.[1]

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

[1] 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

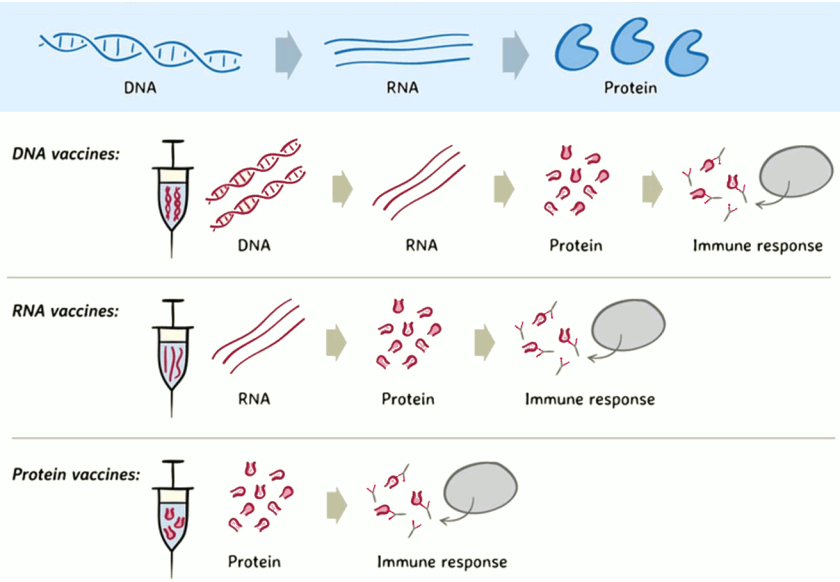
	Method	Example	Risks/drawbacks	Benefits
Conventional	Attenuated live virus	Poliomyelitis (Sabin)	Genetic instability	Lasting immunity
	Chemically inactivated virus	Poliomyelitis (Salk)	Multiple injections	Not infectious
	Recombinant viral proteins	Hepatitis B	Multiple injections	Not infectious
Non-conventional	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.

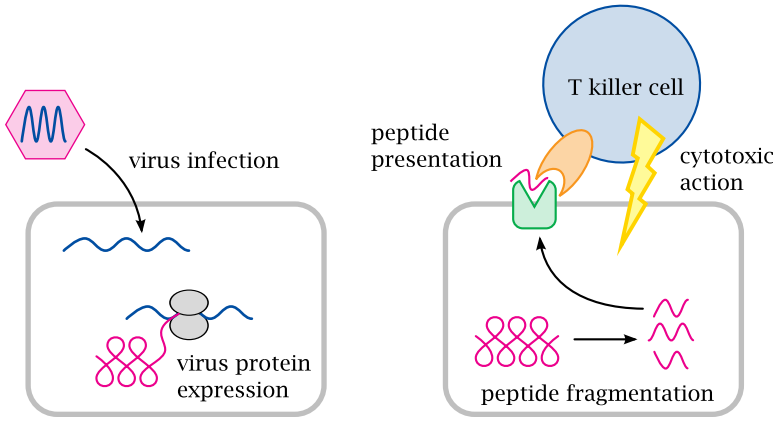
DNA vaccines contain the genes which encode the viral proteins against which we want to induce an immune response; they rely on the cellular machinery for both transcription to mRNA and translation to protein. Such DNA molecules *might* insert themselves into the chromosomal DNA of the host cell.

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

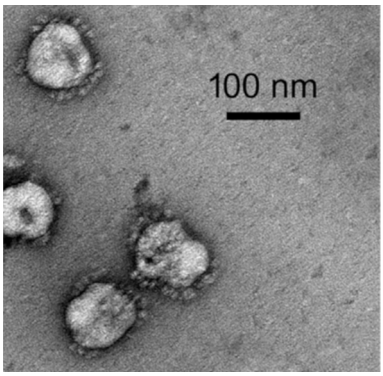
In any viral infection, the body produces not only cytotoxic T-lymphocytes (T-killer cells), but also antibodies. These are extracellular proteins, which will swiftly bind to the surface of the virus particles and thereby prevent them from entering cells

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

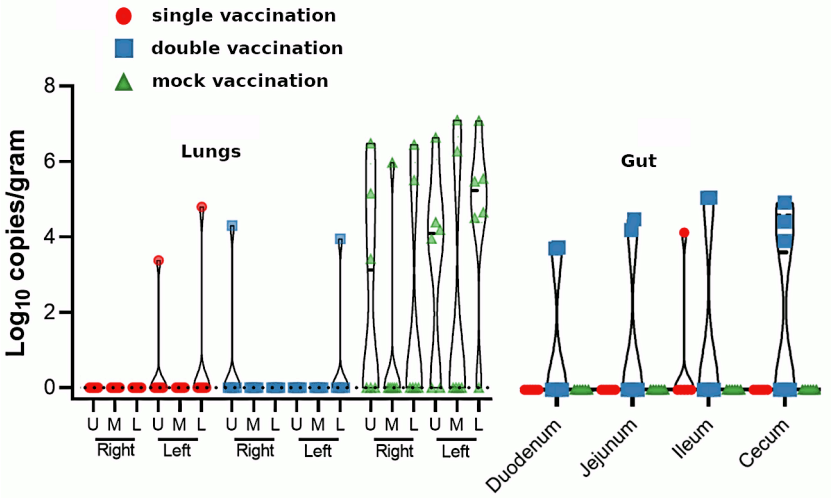
- Vaccine design: gene encoding SARS-CoV-2 spike protein inserted into non-replicating “vector” virus
- Tepid results in animal experiments

- 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 is 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.

Update April 2021: We can note here that the Johnson & Johnson vaccine uses the same methodology as the AstraZeneca vaccine, and both have caused similar adverse effects after the roll-out.

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.[2]

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

[2]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.

not a good idea to tackle a new virus with unproven technology, particularly when time is perceived as being of the essence.

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 1.3.3), 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.

Update April 2021: The Moderna mRNA vaccine has been shown to encode the very same peptide, even though there are some minor differences at the mRNA level. Therefore, until their safety and efficacy profiles are proven different, they can be assumed to be the same.

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.

	Vaccine		Placebo		
	Cases	%	Cases	%	Ratio
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

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.

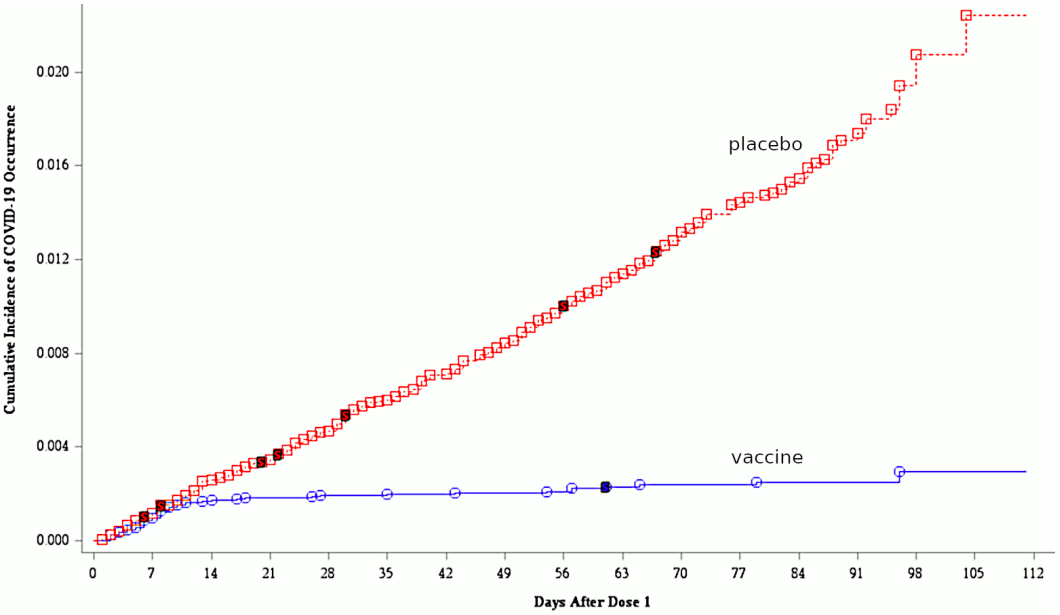
What the FDA memo does not tell us is the number of visits to the doctor that occurred in each group, and the number of PCR tests performed on the patients in question. However, considering the high incidence of flu-like symptoms in the vaccinated group, it is not unlikely that the number of transiently 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

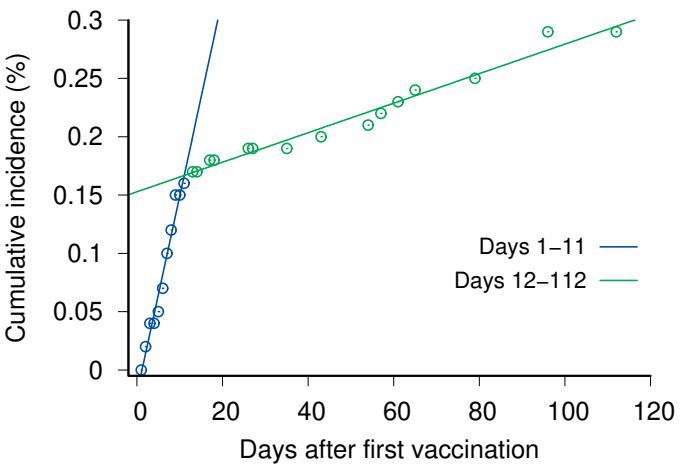
We will now take a closer look at the data contained in the FDA document. The plot shown here is from Figure 2 of the document. The x axis indicates the days after the first injection; the second injection took place between days 19 and 42

(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).

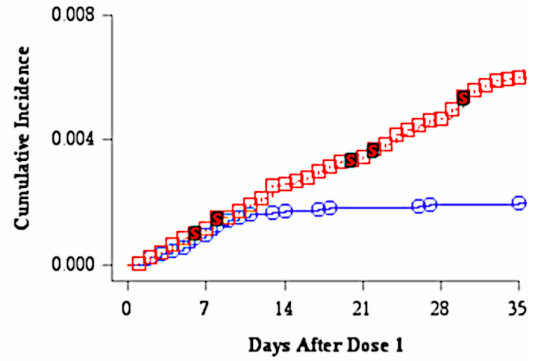
2.5.3 Sudden onset of immunity on day 12 after first injection



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.[3]

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 protocal 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.[4] 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

[3] 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%.

[4] 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—any number of respiratory viruses can cause the same clinical picture.

the FDA should have overlooked such glaring discrepancies proves that the entire review and approval process was a farce.

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	**8**	**0.044**	18325	162	
Previously infected	1767	1		**1847**	**7**	**0.38**

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.

It is interesting to note that India has rejected Pfizer's application for emergency use [22]. Apparently, there are some health authorities still capable and at liberty to make rational and responsible decisions.

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.

2.6 Prevalence of adverse reactions as of spring 2021

As of early April 2021, substantial evidence of adverse reactions to the various vaccines now in use has accumulated.

2.6.1 Adverse reactions to the Pfizer vaccine: initial results from the USA

V-safe Active Surveillance for COVID-19 Vaccines

	Dec 14	Dec 15	Dec 16	Dec 17	Dec 18*
Registrants with recorded 1st 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

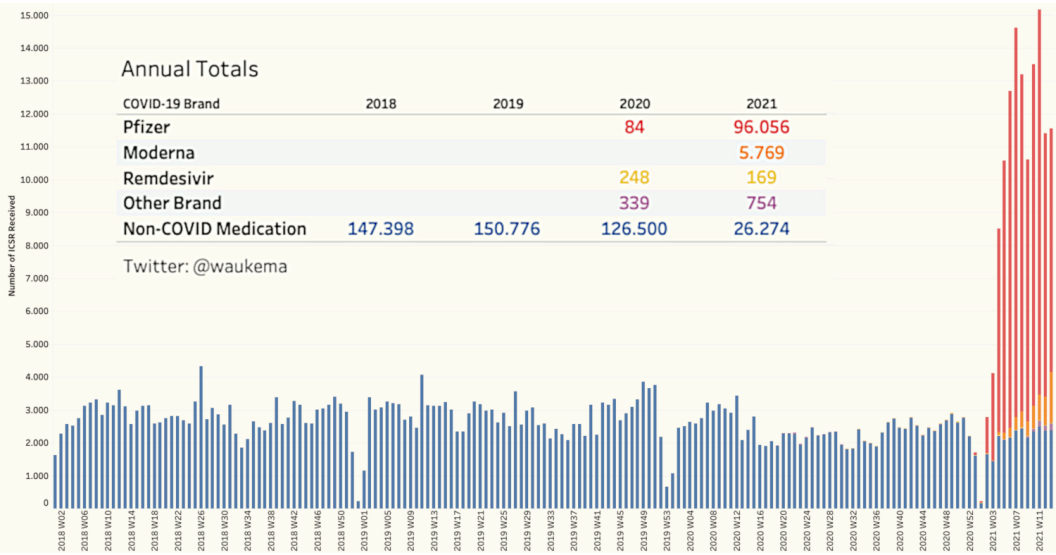
This slide summarizes the frequency of adverse reactions during the first few days of application in the United States. According to this information, which is sourced from the CDC [23], approximately 3% of all vaccinees experienced reactions severe enough to require medical assistance and prevent them 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.

2.6.2 Adverse reactions to vaccines within the European Union

This slide shows the weekly numbers of reports on adverse drug reactions, filed with the European Medicines Agency (EMP). The time series begins in January 2018 and ends with the 11th week of 2021. The graph was produced by Wouter Aukema.

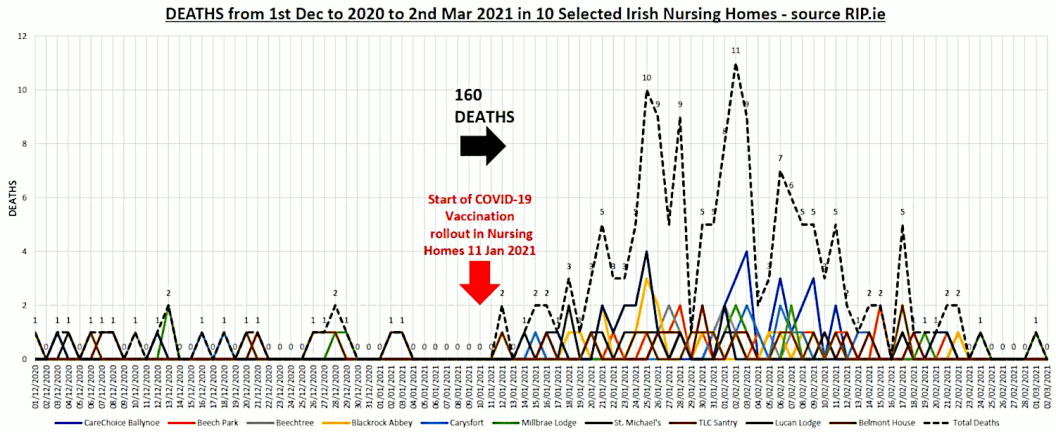
As was the case in the United States, the Pfizer “vaccine” was the first to receive emergency approval by the European Union. It is therefore not surprising to see that the greatest number of adverse events has been reported with this vaccine; however, the Moderna vaccine is ramping up as well. Note that in the year 2021 the Pfizer

vaccine alone received almost four times more reports on adverse events than all non-COVID medications *combined*.



2.6.3 Clusters of deaths after vaccination in senior homes

There are numerous reports—mostly outside the mainstream media, of course—of deaths that occurred in senior homes within weeks of vaccinations. The graphic shown here is from an open letter to Irish College of Physicians [24]; it shows surplus deaths in 10 Irish senior homes that occurred shortly after the onset of vaccinations. (The black dotted line gives the totals, the colored solid lines represent the deaths in individual homes.) A compilation of similar occurrence from various countries has been provided by the UK Medical Freedom Alliance [25].



Another interesting glimpse into the frequency and severity of side effects has been provided by Dr. Charles Hoffe, a family physician from British Columbia, in an

open letter to his provincial government [26]. Hoffe observed 6 cases of severe neurological damage among 900 persons in his clientele who had been given the first shot of the Moderna "vaccine."

We must set such appallingly high rates of adverse effects in relation to the absence of any clearly defined benefit of the vaccines. Against this background, the current push for compulsory vaccination can only be described as criminal—any health officials participating in this campaign must be brought to justice.

2.7 Activation and disruption of blood coagulation

A recurring motif in connection with COVID-19 infections, and likewise with adverse effects of vaccination, is the aberrant activation of blood coagulation. The regular function of blood coagulation is, of course, the emergency repair of ruptured or severed blood vessels in order to staunch the bleeding. The resulting blood clot consists of a meshwork of an insoluble protein called *fibrin*. Enmeshed within this meshwork are the red and white blood cells.

Fibrin is formed from its soluble precursor protein *fibrinogen*, which is abundantly present in the plasma, through the activation of a cascade of proteases. This activation is triggered by specialized proteins, but also by the blood *platelets* (thrombocytes), which recognize the damage to the blood vessels and then stimulate each other to aggregate in place.

Blood coagulation can also be activated by other than mechanical damage to the blood vessels, for example due to autoimmune diseases and infections.

2.7.1 Manifestations of disrupted blood coagulation

1. blood clot formation in the veins (venous thrombosis),
2. blood clot formation in the arteries,
3. consumption of plasma coagulation factors and blood platelets by *disseminated intravascular coagulation*.

Venous thrombosis preferentially affects anatomical locations in which blood flow is rather slow, such as some major veins inside the skull. This so-called cerebral venous sinus thrombosis is a very serious disease, often deadly. It is normally extremely rare; its repeated occurrence after injection with the two adenovirus-based vaccines manufactured by AstraZeneca [27] and also and Johnson & Johnson was impossible to ignore even in the current climate of recklessness and hysteria, and it led to suspensions of these two vaccines in certain jurisdictions.

Disseminated intravascular coagulation (DIC) normally occurs only in severe trauma or infection. If a systemic infection spreads through the bloodstream, damaging to the blood vessels and setting off blood coagulation simultaneously in many locations, this may use up the blood platelets as well as fibrinogen and the other plasmatic coagulation factors. Thus, in addition to impeded blood flow in interior organs caused by those ubiquitous blood clots, there will be bleeding. Needless to

say, DIC also is a highly dangerous condition, and its occurrence after vaccination, in the absence of any other underlying disease [27], likewise was impossible to ignore.

The increased formation of fibrin will also set off its increased breakdown. Increased blood levels of one of the breakdown products, the so-called *D-dimers*, are used diagnostically to detect DIC.

Blood clot formation in the arteries may result in e.g. heart attack and stroke. These are rather common in the general population at any time; therefore, even substantial additional incidence of such events after infection or vaccination may well go unnoticed.

2.7.2 The role of the spike protein

- The ACE2 receptor is expressed on blood platelets [28]
- The alternate receptor CLEC4M (CD209L) is expressed on endothelial cells
- The spike protein as such can directly activate platelets, and cell-attached spike protein it may direct the immune system against endothelial cells and platelets

We had discussed above that the SARS-CoV-2 spike protein has been engineered to recognize two different receptors (see Section 1.3). There is apparently a third one (CD209), which is similar to CLEC4M (CD209L) and also binds the spike protein [11]. Between them, these receptors permit the virus to bind and enter a considerable range of cell types.

The cell types most relevant to blood coagulation are the platelets and the endothelial cells, that is, the innermost cell layer of the blood vessels which is in direct contact with the blood. Platelets may be directly activated by bound spike protein. Endothelial cells may be damaged by the immune system if they are infected by the virus. This may involve the T killer cells (see Section 2.2.2); alternatively, it may involve antibodies, which activate the so-called *complement system*, a cascade of cytotoxic plasma proteins. The most important target for the antibodies will again be the spike protein.

2.7.3 Blood coagulation disorders due to the virus infection itself

- Histopathology shows damage and thrombosis of small blood vessels in the lungs [29]
- Case reports of immune thrombocytopenia in COVID-19 patients [30, 31]
- Association of thrombocytopenia with severe disease [32]
- Elevated D-dimer levels, recommendation of therapeutic anticoagulation [33]

The studies summarized here show that coagulation disorders are common in severe cases of COVID. Countermeasures include direct inhibition of coagulation with heparin and suppression of the underlying inflammation with corticosteroids (e.g. dexamethasone or budesonide).

2.7.4 Blood coagulation disorders due to vaccination

- Case report of jugular vein thrombophlebitis [34] after Pfizer vaccine
- Case reports of ITP 2 days after Moderna vaccine [35] and 3 days after Pfizer vaccine [36]
- Review of reported cases of immune thrombocytopenia—assuming that all cases are reported, the CDC collection does not indicate increased rate after vaccination [37]
- Statistically robust increase of rare coagulation disorders after AstraZeneca vaccine [27]

We see that the disorders after vaccination resemble those after infection with the virus. This is of course expected, given that the vaccines induce the biosynthesis of the viral spike protein—the very molecule that is at the heart of disrupted coagulation. Focusing only on this particularly noxious protein for immunization surely qualifies as one of the worst ideas in the history of vaccine development.

We may wonder why disrupted coagulation is severe in a few patients, whereas the majority seems unaffected by it. One possible explanation is that in affected individuals, through mere chance, a blood vessel is punctured by the intramuscular injection, which causes a substantial amount of the vaccine to enter the circulation directly. Once in the bloodstream, the "vaccine" particles enter the vascular endothelial cells, which then start expressing the viral spike protein.

Even without direct injection into the bloodstream, the vaccine may enter the circulation after passage through the lymphatics. We currently do not know for certain to what extent the transport route determines the occurrence and severity of adverse effects related to blood clotting.

2.8 Immediate allergic reactions

These have been acknowledged officially, and patients with prior severe allergy to other substances have been warned against getting vaccinated, or to receive their vaccine shots in a facility that is equipped for emergency medical assistance. The allergic reactions take the form of acute circulatory collapse or shock within minutes after the injection. It is most likely due not to the viral genes (mRNA) but to the mix of synthetic lipids and other compounds used for "packaging" the mRNA and promoting its uptake into cells. One candidate compound is polyethyleneglycol (PEG) [38], allergies to which are known from other pharmaceutical uses (see e.g. [39, 40]). However, the lipid components may well play an important role as well; they are simply too new at this point for us to be certain either way.

Chapter 3

The Pfizer mRNA vaccine: pharmacokinetics and toxicity

3.1 Introduction and background

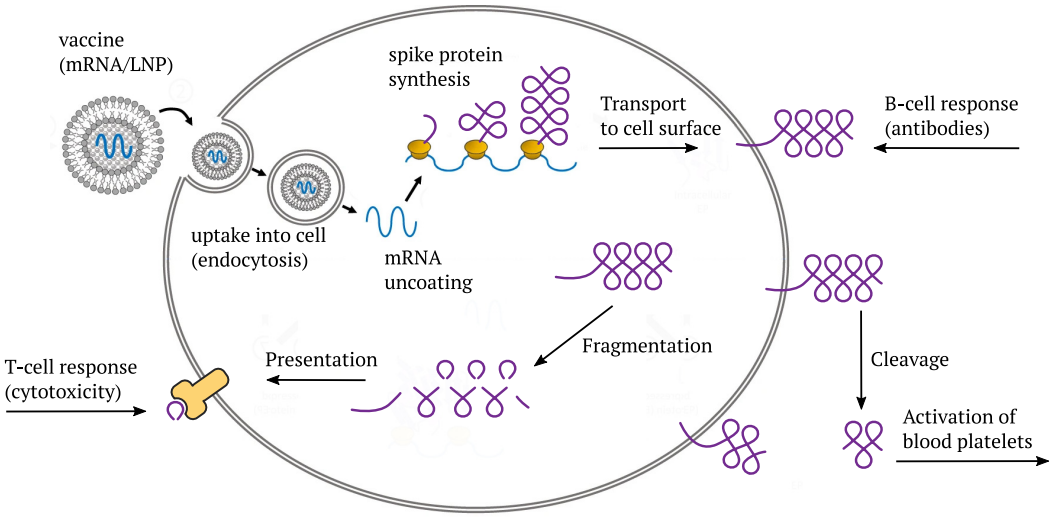
As with any drug, a key consideration for the toxicity of the COVID mRNA vaccines is where exactly in the body they end up, and for how long they will stay there. Such questions are the subject of *pharmacokinetics*; and during drug development, they are usually thoroughly investigated, at first in animal experiments and subsequently in a small number of human volunteers. Only after such preliminary studies have been successfully concluded will proper clinical trials be approved, which will then determine whether the drug or vaccine in question has the desired clinical efficacy.

Because of the officially sanctioned haste and systematic gross negligence in the development and approval of the COVID-19 vaccines, our knowledge of their pharmacokinetics is sketchy. The only somewhat detailed animal study that has reached the public pertains to the Pfizer vaccine. These data were publicized after Pfizer had filed them with the Health authorities in Japan when applying for emergency use authorization of its vaccine in that country.[1] These data pertained in particular to the distribution of the vaccine within the body after injection and to its elimination from the body. Even though far from being comprehensive or even adequate, this document has rather far-reaching implications: it shows that Pfizer—as well as the authorities that were apprised of these data— must have recognized the grave risks of adverse events after vaccination even before the onset of clinical trials. Nevertheless, Pfizer's own clinical trials failed to monitor any of the clinical risks that were clearly evident from these data.

Before we discuss this study and its implications in detail, we will briefly review how the Pfizer mRNA vaccine works. These explanations also apply to the Moderna mRNA vaccine, whereas the AstraZeneca and the Johnson & Johnson vaccines differ in some aspects.

[1] The same data may also have been filed in the U.S. and other wester countries, but the FDA and the corresponding health regulators did not release them.

3.1.1 How the mRNA COVID vaccines work



The Pfizer and Moderna mRNA vaccines consist of a synthetic messenger RNA (mRNA) that encodes the SARS-CoV-2 "spike protein," which normally is found on the surface of the coronavirus particles. This mRNA is coated with a mixture of synthetic *lipids* (fat-like molecules) that protect it during transport within the body, and which also facilitate its uptake into the target cells through *endocytosis*.

After the vaccine has entered a cell, it initially finds itself enclosed by a membrane vesicle—a little bubble that was pinched off from the cell membrane. The subsequent accumulation of acid inside this bubble causes the lipids to be stripped off, and the mRNA to be released into the cytosol (the intracellular fluid). It then binds to *ribosomes*—the cell's little protein factories—and induces the synthesis of the actual spike protein molecules. Most of the spike protein molecules will then be transported to the cell surface.

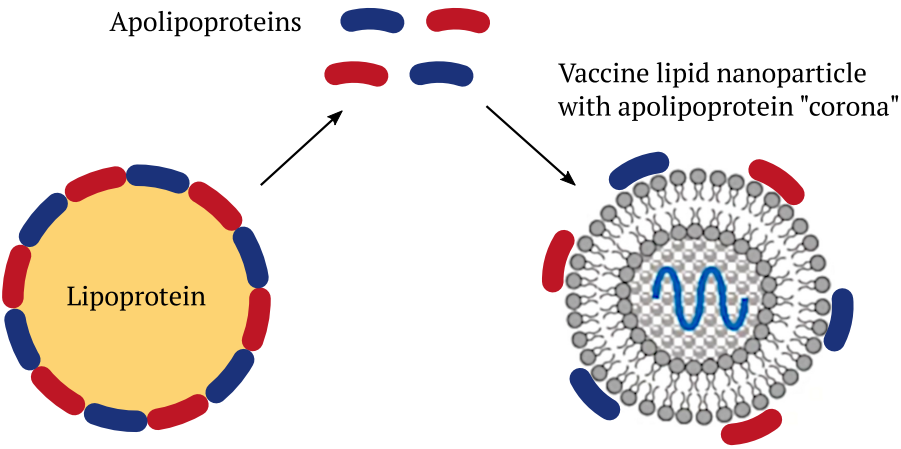
Once it appears there, it will be recognized by B-lymphocytes (B-cells), which will then start making antibodies to it. Furthermore, some part of the spike can also be cleaved off by proteases on the cell surface and released from the cell. If this happens within the circulation, the released fragment—referred to as *S1*—can bind to blood platelets (thrombocytes) and activate them. This promotes blood clotting.

As with any protein that is synthesized within the cell, a small number of molecules will undergo fragmentation, and the fragments will be presented on the cell surface in association with specific (HLA-) carrier proteins. The purpose of this mechanism is immune surveillance—as soon as fragments show up of some protein which the immune system does not recognize as "self," an immune response will be mounted against it and against the cells that produce this protein. This response is mediated by cytotoxic T-lymphocytes (CTLs, T-killer cells).

In mounting its cytotoxic response, the immune system will not distinguish between a true virus infection and the expression of an mRNA vaccine—as long as the spike protein fragments appear on the cell, the killer cells will be on the march. If the vaccine is expressed in the cells that line the blood vessels—the *endothelial*

cells—the vascular lesion caused by the immune attack will once more set off blood clotting. Thus, we have at least two distinct paths toward blood clotting after vaccination.

3.1.2 The lipid-coated mRNA vaccines acquire an apolipoprotein “corona”



Lipoprotein particles occur naturally in the bloodstream and within the tissues of our body. They consist of a core of lipids and are surrounded with a shell of proteins called *apolipoproteins*. Their purpose is to transport lipids such as cholesterol and triacylglycerol (regular fat) between organs. For example, a specific type of lipoprotein called *chylomicrons* transports dietary fats after they have been taken up in the small intestine, and lipoproteins called VLDL and LDL distribute fats that have been synthesized in the liver to other organs and tissues.

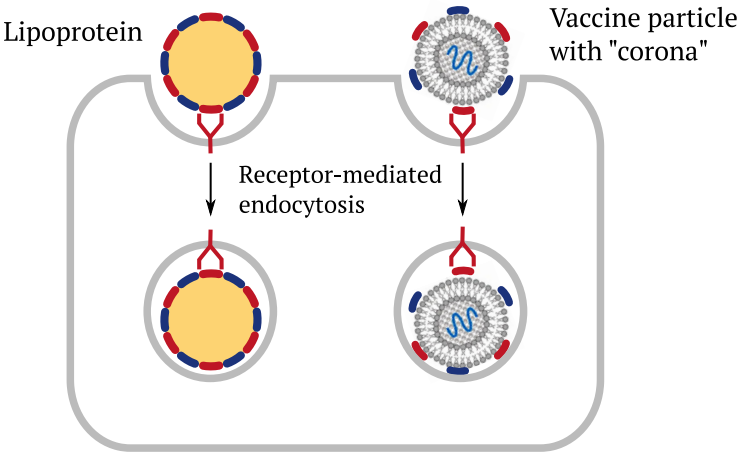
The various apolipoproteins that encase the lipoproteins stabilize the particles, and they also serve as “address tags” that bind to receptor molecules on cell surfaces; this interaction will trigger the uptake of the lipoproteins into those cells. Artificial lipid nanoparticles (LNPs) like those used in the COVID mRNA vaccines can acquire a shell—a “corona”—of the body’s own apolipoprotein molecules [41]. This enables these vaccines to be taken up into the cells of our body, too.

The liver has a central place in lipid and lipoprotein metabolic turnover. Accordingly, liver cells are rich in specific surface receptor molecules which mediate lipoprotein uptake, suggesting that they will efficiently take up LNPs decorated with apolipoproteins also. This is indeed the case. However, other organs have high rates of lipoprotein uptake, too, and they must therefore be expected to accumulate the apolipoprotein-decorated vaccine LNPs as well.

3.1.3 Receptor-mediated cellular uptake of lipoproteins and of vaccines

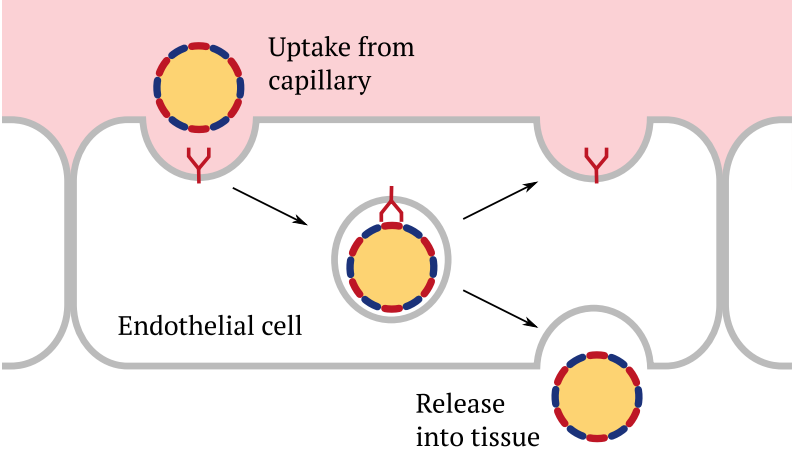
This slide illustrates the role of cellular receptors and the apolipoproteins in facilitating the uptake of vaccines into cells through endocytosis. They bind to the same cellular receptors as the regular lipoprotein particles do, and they subsequently get

taken up in the same manner. The subsequent events—release of the mRNA and protein synthesis—have already been discussed above.



3.1.4 Transcytosis of lipoproteins from the bloodstream into the tissues

All substrate exchange between the tissues and the bloodstream occurs in the capillaries. In these finest of all blood vessels, the blood is separated from the extracellular matrix of the tissues by only one cellular layer—namely, the endothelial cells. The capillary wall permits free passage only to small molecules such as for example blood sugar (glucose) or amino acids. The lipoproteins, which are far larger, must be transported across the capillary wall by *transcytosis*. In this two-stage process, endocytosis on one side of the cell is followed by *exocytosis*, that is, by release of the particles, which occurs on the other side.



While this figure shows transcytosis from the bloodstream to the tissue, the process actually works in both directions. In this manner, cells in the tissues can avail

themselves of cholesterol carried by circulating LDL, but they can also return surplus cholesterol through the bloodstream to the liver via other lipoproteins (HDL).

Transcytosis will also apply to the "corona"-decorated vaccine LNPs and enable them to reach the tissues in various organs. Reverse transcytosis of vaccine might contribute to its uptake from the muscle tissue into the circulation after injection.

3.2 The Pfizer vaccine pharmacokinetics study on rats

- A "model vaccine" was used—same LNPs, different mRNA (coding for luciferase)
- Cholesterol contained in the LNPs was labeled with radioactivity (^{3}H) for tracing
- The distribution of the lipid between different organs was measured at various times following intramuscular injection

This is the key experiment in the study which was submitted by Pfizer to the Japanese authorities [42]. The technical approach used here is quite common, since radioactivity can be very sensitively and accurately measured. The radioactively labeled vaccine preparation was injected into animals (rats). The animals were "sacrificed" (cut up) at various time points after the injection, and the amount of radioactivity in different organs was measured.

The model protein used in this study was a firefly protein called *luciferase*. This is the very protein that permits fireflies to glow in the dark. When the rats' body cells take up the mRNA that encodes luciferase and then synthesize the protein, they, too, will begin to glow in the dark. Light, like radioactivity, is convenient to measure; the more light that emanates from a given tissue, the more mRNA uptake and protein synthesis have occurred.[2] Therefore, between the radiolabel on the lipid and the luminescence elicited by luciferase, it was possible to determine both the distribution of the model vaccine within the body and its biological activity.

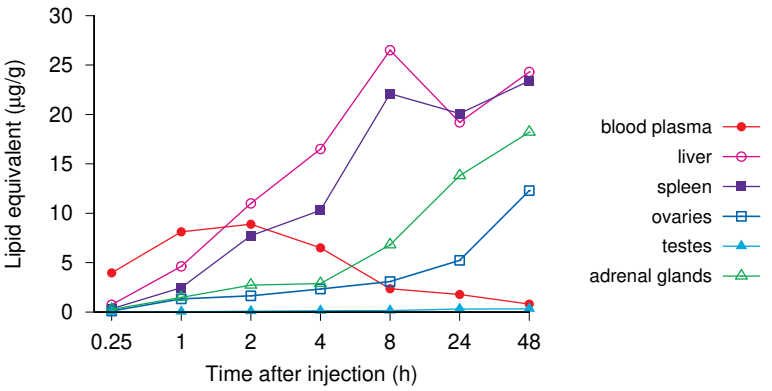
3.2.1 Key data from the lipid distribution study

The first thing to note is that the labeled lipid shows up in the blood plasma after a very short time. The highest plasma level is reached at two hours after the injection; however, even after only 15 minutes (0.25 hours) the level already reaches almost half of that maximal value. This rapid uptake might occur for example through reverse transcytosis into the capillaries (see above), or through accelerated lymphatic drainage downstream of the acute release of inflammatory mediators.

As the blood plasma level drops off, the activity rises in several other organs. The fastest and highest rise is observed in the liver and the spleen. Both of these organs are rich in *macrophages*, a cell type that is in charge of clearing particles such as microbes or the fragments of decayed cells from the bloodstream. Macrophages

[2] To generate light, luciferase also requires a specific small-molecule substrate named luciferin and adenosine triphosphate (ATP). The luminescence assay is therefore more complex and less quantitatively accurate than measurements of radioactivity.

are also numerous in the bone marrow, where the vaccine reaches somewhat lower but still substantial levels (not shown).



While the macrophages are likely responsible for most of the uptake in the spleen, this may not be the case in the liver. Here, the vaccine likely ends up mostly in the organ-specific epithelial cells, which are very rich in lipoprotein receptors.

Uptake into the ovaries and into the adrenal glands is most likely also mediated by lipoprotein receptors. Both organs take up lipoproteins to obtain cholesterol, which they use as a precursor for producing steroid hormones—corticosteroids in the adrenal glands, and female sexual hormones (estrogens and progestins) in the ovaries. The testes, too, produce sexual hormones (in particular testosterone) from cholesterol, but here the accumulation of vaccine lipid is remarkably is much lower. The scientific literature does not offer a full, straightforward explanation, but the restricted uptake into the testes may be related to the so-called blood-testes-barrier.

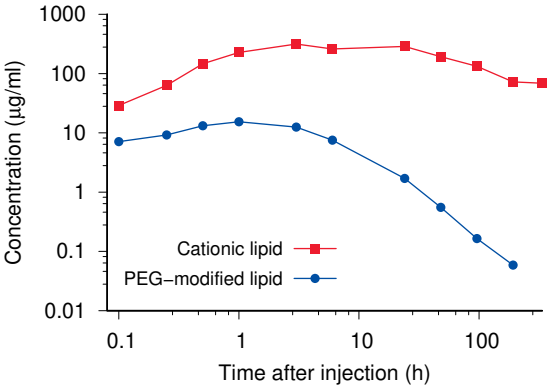
In most other organs which were examined the levels were similarly low as in the testes. We note, however, that at least the blood vessels within all organs will be affected in every organ and in every tissue.

It is noteworthy that the level of radioactivity rises fastest in the liver but then stagnates, whereas in the ovaries and the adrenal glands the rise continues unabated even two full days after the injection. This suggests that the radioactivity may be redistributed from the liver to the glands. We must remember that the LNP component which carried the label is cholesterol. The labeled cholesterol would behave just like endogenous (unlabeled) cholesterol, and we would expect it to be recycled and redistributed after uptake in the liver. To what extent the *other* lipids contained in the LNPs would undergo such redistribution and also accumulate e.g. in the ovaries is an open question that could only be answered by experiment—namely by radiolabeling each of them in turn. It should go without saying that the use of the vaccine should never have been authorized without studies of this kind.

It must also be noted that the distribution of the vaccine might be affected by the protein encoded by its mRNA component. If instead of the presumably inert luciferase enzyme the spike protein had been expressed, this might have affected vascular integrity, particularly also at the blood brain barrier. It should go without saying that the FDA, the EMA and other regulators should have obligated Pfizer

to conduct thorough studies on the disposal of the non-natural lipids and on the pharmacokinetics actual vaccine before considering its authorization for clinical use.

3.2.2 Very slow elimination of the cationic lipid ALC-0315 from rat liver



In addition to cholesterol, the LNPs contain another naturally occuring lipid (distearoyl-phosphatidylcholine) and two non-natural ones. One of these (ALC-0315) is weakly basic, whereas the other (ALC-0159) carries a polyethyleneglycol (PEG) moiety. With these two non-natural lipids, Pfizer did report the change over time of their concentrations within the liver. The level of one lipid dropped slowly but regularly with time. The other one, however—the cationic lipid ALC-0315—remained at very high levels even two weeks (336 hours) after the injection. Even after 6 weeks some of the compound was still detected in liver.

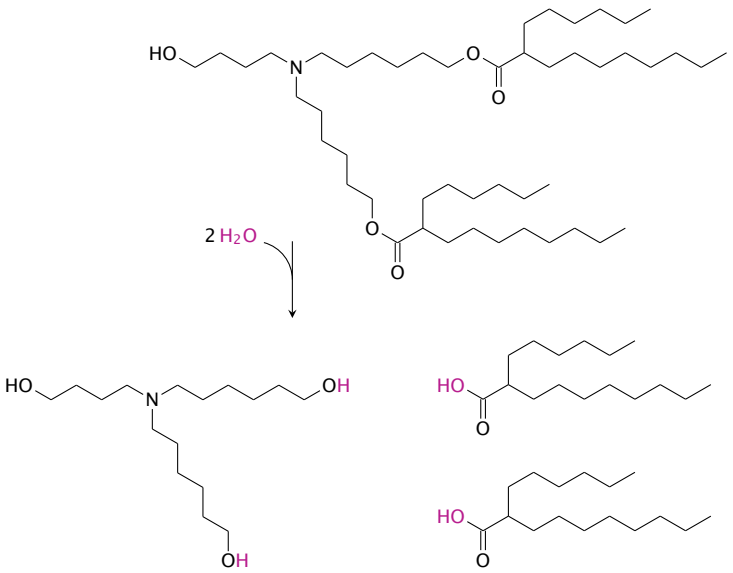
As discussed in the preceding section, we cannot rule out that these synthetic lipids, too, are redistributed from the liver to other organs, where they might then be stored for even longer periods of time. You may have heard that some pesticides such as DDT can persist in the human body for months and even years. This typically occurs with compounds which are very *lipophilic*, meaning that they partition into fat droplets within fat tissue and other organs. As long as the fat within these droplets is not utilized, the chemicals dissolved within them will be safe from metabolic turnover and degradation. The cationic lipid ALC-0315 is likely able to accumulate in the same manner. If so, we can expect persistence for even longer periods of time than evident from this graph in tissues that have lower metabolic activity than the liver.

3.2.3 Slow degradation is built into the structure of ALC-0315

This topic is rather technical, and it is not necessary for the big picture. If you can't make out what this diagram is showing, feel free to skip it.

The structure at the top shows the intact cationic lipid referred to as ALC-0315. Hydrolysis of the two ester (C(=O)O) bonds produces the three fragments at the bottom; according to Pfizer's document, this is the initial step in the degradation

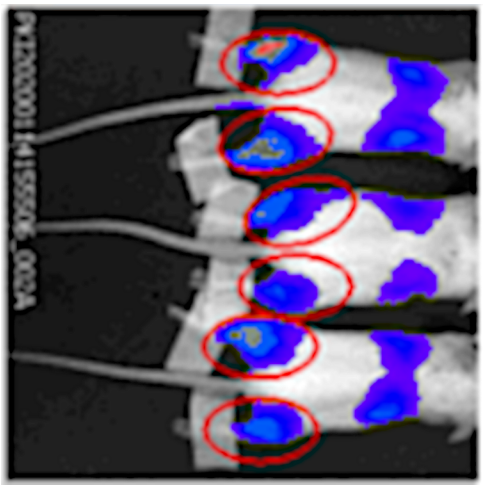
and elimination pathway of this lipid. The following features suggest that inside the body this step will occur rather slowly:



1. The entire molecule contains no permanent charge and only one ionizable atom (the nitrogen, N), which is linked to three alkyl chains. Aside from the one polar hydroxy (OH) group, the entire remainder of the molecule is hydrophobic. This means that the molecule will partition very strongly not only into lipid bilayers (membranes) but also into lipid droplets, where it will be effectively hidden from any degradative enzymes.
2. When this molecule is part of a lipid bilayer, as is the case within the vaccine LNPs, the two ester bonds will still be buried deep within the hydrophobic portion of that bilayer, which will protect them from hydrolytic cleavage.
3. Hydrolysis of the ester bonds will to some degree be sterically hindered by the the adjacent branches in the fatty acyl residues.

With the possible exception of the lack of a permanent charge, none of these features is essential for the desired function of the molecule, namely to release the mRNA from the vaccine particles after the latter have been taken up into our body cells. There are many ways in which this molecule could have been modified for faster degradation in vivo. It is noteworthy that this was not done—the vaccine was deliberately formulated with a compound that is degraded and eliminated from the body very slowly. Given that this lipid will most likely stay in our tissues for months, we must expect *cumulative toxicity* with repeated vaccinations.

3.2.4 Strong expression of luciferase in the rat liver and spleen



This picture is taken from the Pfizer study. As far as I can tell, it shows three skinned rat bodies. The time point of the measurement is 6 hours after the injection. The red ovals indicate the injection sites in the hind legs, and the various colors (mostly blue) within them indicate the luminescence produced by the local expression of luciferase. This luminescence indicates that the vaccine entered cells near the injection sites and successfully delivered its mRNA to the ribosomes within the cell.

The separate blue and purple areas to the right are over the liver and the spleen. Thus, the pronounced accumulation of lipid in these organs correlates with strong expression of the delivered luciferase mRNA also.

3.2.5 Does the correlation between lipid uptake and mRNA expression apply to other organs, too?

- Luciferase expression could have been tested with other organs, but no such results were reported
- Distribution of mRNA could easily have been traced directly
- Lipids and mRNA are tightly bound to each other until after they are taken up into cells, suggesting that the correlation holds

It would of course have been important to study the expression of the mRNA in other organs also, particularly those that took up large amounts of the LNPs, such as the ovaries. Rat ovaries are small, and therefore luminescence measurements might not be very sensitive; however, in that case, such measurements could have been performed on a larger animal species.

At the very least, if expression analysis was deemed too cumbersome, it would have been easy enough to detect the uptake of the mRNA itself into different tissues, for example by labeling it with radioactive iodine [43]. Such measurements would

have been even more accurate and straightforward than those which were actually carried out for the lipids.

Since these experiments would not have been particularly difficult, I suspect that Pfizer did in fact perform them, but decided not to report the results. Be that as it may, however—we do know that most of the lipid will remain bound to the mRNA until after both have been taken up into cells. In the absence of proof positive to the opposite, we must therefore assume that a close correlation exists between lipid uptake, mRNA uptake, and mRNA expression. This raises obvious concerns for the health and integrity of the ovaries.

3.3 What do Pfizer's animal data signify for biological effects in humans?

- Rapid appearance of spike protein in the circulation
- Toxicity to organs with observed high rates of uptake
- Toxicity to organs with *expected* high rates of uptake, in particular placenta and lactating breast glands
- Penetration of some organs might be higher with the real vaccine than with this luciferase model

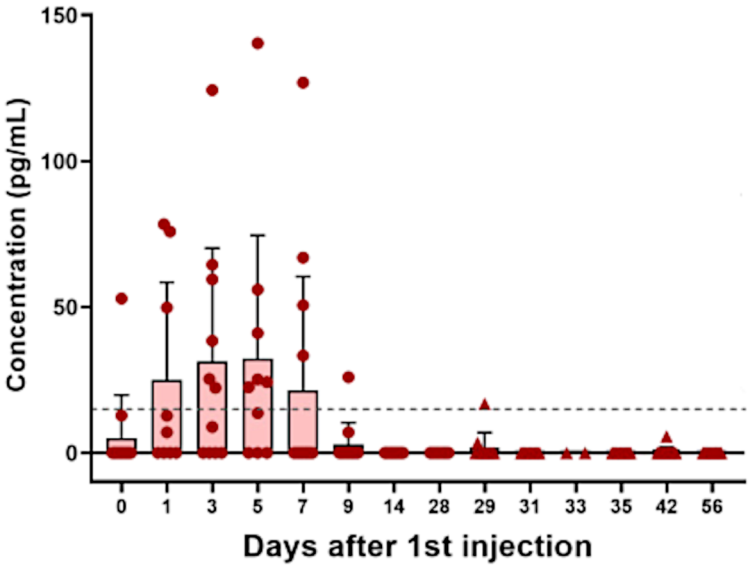
The rapid entry of the model vaccine into the circulation means that we must expect that spike protein will be expressed within the circulation, particularly by endothelial cells. We have seen before that this will lead to activation of blood clotting through direct activation of platelets and also, probably more importantly, through immune attack on the endothelial cells.

We must furthermore expect damage to organs that take up high amounts of the vaccine. This is in fact documented in the EMA assessment report on the Pfizer vaccine [44] for the muscle tissue at the injection site (p. 49) and also for the liver (p. 46).

Moreover, we must expect toxicity to some organs that were not examined in Pfizer's study. This includes in particular the placenta, which like the ovaries produces large amounts of progestin hormones from cholesterol, likewise acquired from circulating lipoproteins, and the lactating mammary glands, which acquire fat and cholesterol contained in lipoproteins for secretion into the breast milk.

While the distribution studies discussed here did provide some useful and relevant information, we must note that the expression of the spike protein instead of the presumably inert luciferase enzyme might affect the distribution of the vaccine due to its interference with vascular integrity, including at the blood brain barrier. The actual COVID vaccine might therefore achieve greater entry into the brain than the luciferase model vaccine. The FDA, the EMA and other regulators should have insisted that such experiments be carried out and documented.

3.3.1 Expression of spike protein shortly after injection of an mRNA vaccine into humans



The early entry of the vaccine into the circulation observed in animals leads us to expect the same in humans. In keeping with this, spike protein becomes detectable in the blood plasma of human vaccinees even on the day of the injection (day 0) and peaks several days later [45]. Note that this assay measured only the S1 fragment that was cleaved from the cell surface and released, not the intact spike that remained on the cells (see Section 3.1.1).

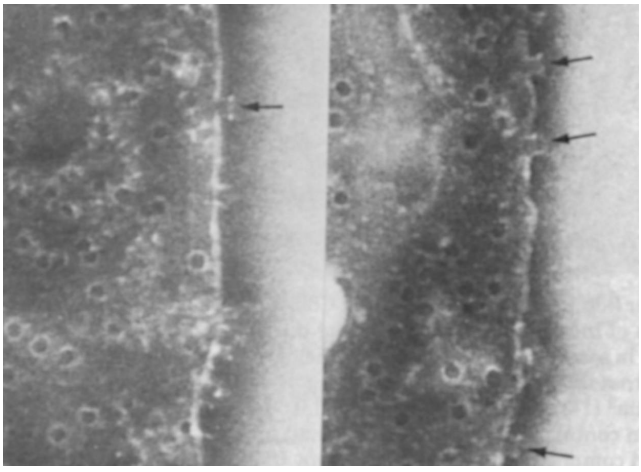
The triangles on day 29 and later in the above figure indicate the levels of free S1 fragment after the second injection. The very low levels most likely do not reflect a failure of the injected mRNA to be expressed, but rather result from the immune response triggered by the first injection. Circulating antibodies will bind to the spike protein and interfere with its measurement. The resulting spike-antibody complexes may be cleared from the bloodstream by phagocytes, but they may also contribute to inflammation. The same antibodies would also bind to the spike protein that remains on the cells. Once bound, they can set off the *complement system*, a cascade of plasma proteins that ultimately kills cells by punching holes into them [46].

Together with rising antibodies, the first injection will also induce T-killer cells directed against the spike-producing cells (see Section 3.1.1). The more rapid and intense cytotoxic action of these T-cells may destroy the cells which took up the vaccine before they had much time to produce spike protein. Whatever the relative contributions of antibodies/complement and of cytotoxic T-cells to the suppression of free spike protein levels after the second injection may be, it is clear that this finding indicates greater harm to the blood vessels than after the first injection.

It deserves mention that the above data were obtained from a fairly small sample—13 persons overall, out of whom 11 exhibited detectable free S1 fragment. Quite possibly, even higher levels would have been observed among a larger group

of test persons. Altogether, the findings in this study substantiate the hypothetical mechanism of vaccine-induced blood clotting that was stated clearly and very early on by the Doctors for Covid Ethics [47], and which has since been fully borne out by experience [48].

3.3.2 Complement pores on the surfaces of red blood cells



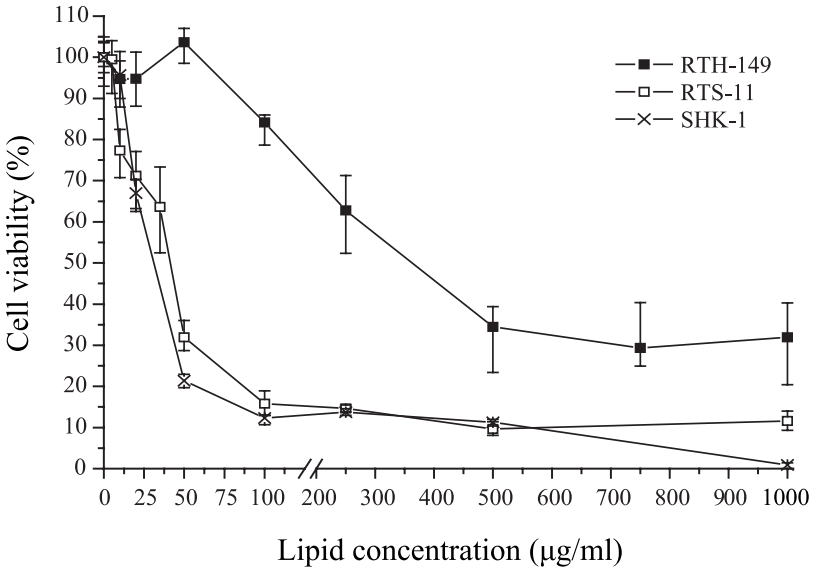
We just saw that in particular the second injection will likely cause the activation of the complement system on endothelial cells. This picture, which is taken from a seminal paper on the action mode of the complement system [46], illustrates that the complement system is perfectly capable of "shooting up the joint"—of utterly destroying a cell.

In the depicted experiment, antibodies against sheep red blood cells were allowed to bind to such cells in the presence of human serum, which provided the complement proteins [49]. As you can see, the cells are riddled with holes. An individual pore consists of multiple complement protein molecules; it protrudes from the membrane (see arrows) and has a diameter of approximately 10 nanometers. The pores will break down the barrier function of the cell membrane, and the cell will die.

Similar effects must be expected with endothelial cells downstream of spike protein expression and antibody binding. The damage to the capillaries will promote vascular leakage as well as blood clotting.

3.3.3 Cationic lipids are cytotoxic

This graph is taken from a study [50] that is not related to the Pfizer vaccine; it is intended only as an illustration of cationic lipid toxicity in general. It shows the dose-dependent effect of the cationic lipid in question (stearylamine) on the viability of three different cell lines. Among these, the two macrophage-like lines RTS-11 and SHK-1 are more sensitive to the cytotoxic effect than the liver-derived cell line RTH-149.



While the various cationic lipids that have been used for DNA or mRNA delivery differ in cytotoxicity, they all are toxic to some degree; and as this figure illustrates, various cell types differ in susceptibility. The high susceptibility of macrophages is due to their built-in capability to produce reactive oxygen species (ROS) such as hydrogen peroxide and superoxide. When this pathway is triggered by cationic lipids, the ROS produced may kill the cells outright—as observed in the depicted experiment. A lower level of activation may cause the macrophages to "misbehave," which may lead to inflammation, autoimmune disease, and potentially cancer.

It is interesting to note that the above-mentioned evidence of liver and muscle toxicity in the EMA report was obtained with the model mRNA encoding the presumably non-toxic luciferase enzyme. Therefore, this observed toxicity does not involve the spike protein. Luciferase, unlike spike protein, is not transported to the cell surface; and moreover the animals will have had no pre-existing immunity to luciferase that could have set off a rapid, intense immune response. We thus infer that the reported cell damage is due to chemical toxicity, mediated most likely by the cationic lipid components of the LNPs. Accordingly, future vaccines that use the same delivery technology must be expected to share this toxicity, regardless of whether they be directed against the spike protein, another SARS-CoV-2 antigen, or a different disease altogether.

3.3.4 Toxicity in tissues and organs

- Muscle fiber degeneration and scarring
- Subcutaneous inflammation
- Liver cell vacuolization and degeneration
- Inflammation and function damage to nerves and joints

These findings from rat experiments are listed in the [44]. They, too, were obtained using the model vaccine which coded for luciferase rather than the actual SARS-CoV-2 spike protein, which means that the toxicity is most likely due to the cationic lipids in the LNPs. It must be noted that none of these toxic effects observed in animals were monitored in the so-called clinical trials. They do, however, correspond to adverse effects observed in vaccinees since the onset of mass vaccinations.

3.3.5 Animal data on reproductive toxicity

- Very limited data collected in only one animal species (rats)
- Loss of early embryos before implantation in the uterus >2 times more common in vaccine group than in controls
- Malformations more common in vaccine group than in controls

Pfizer tested its vaccine for reproductive toxicity on only one species (rats) and on only small numbers of animals (21 litters). A greater than twofold increase in pre-implantation loss of embryos was noted, with a rate of 9.77% in the vaccine group, compared to 4.09% in the control group. The EMA report merely states that the higher value was "within historical control data range" [44, p. 50]. EMA should of course have obliged Pfizer to state unambiguously whether or not the observed difference was statistically significant; and if it was not, to increase sample sizes so as to ensure the required statistical power.

The same criticism applies to the reported observations of "very low incidence of gastroschisis, mouth/jaw malformations, right sided aortic arch, and cervical vertebrae abnormalities." Overall, Pfizer's studies are inadequately described and apparently were also inadequately carried out.

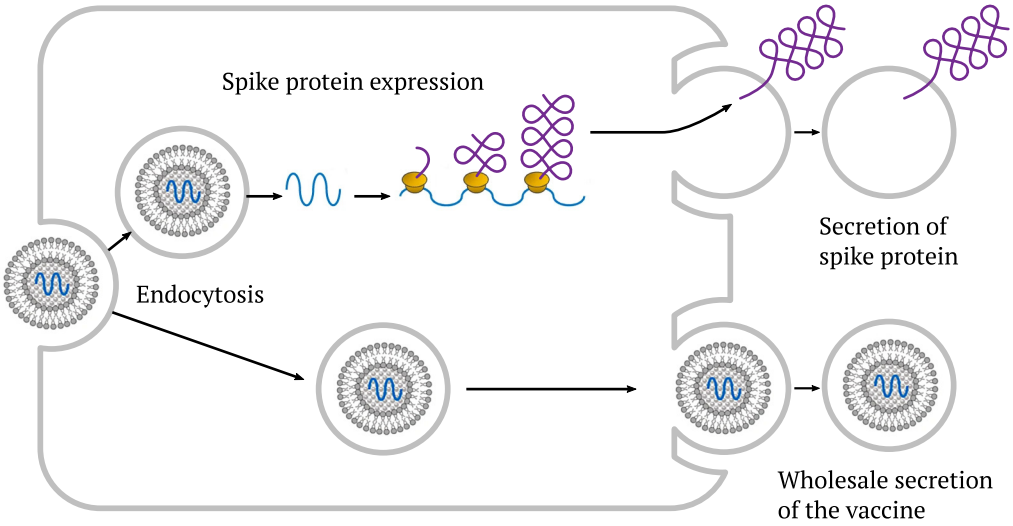
The observed pre-implantation loss indicates toxicity at a very early stage of development, either to the embryo or the nascent placenta. It might be caused by a high level of spike protein expression, but also by toxic lipids; and it might occur already within the ovaries, but also affect the fertilized egg or subsequent developmental stages within the Fallopian tubes or the uterus. This also applies to malformations, although these would more likely be caused by damage later on in embryonic development, suggesting transfer of toxicity across the placenta.

3.3.6 Two possible pathways for vaccine toxicity to breastfed infants

Uptake of the vaccine by mammary gland cells opens two possible pathways of toxicity to the breastfed child: firstly, the expression of spike protein and its secretion into the breast milk, and secondly, the wholesale transfer of the vaccine into the milk.

The mammary glands are *apocrine*, which means that they pinch off and release fragments of their own cytoplasm into the milk; thus, anything that has reached the cytoplasm might also reach the breast milk. In this connection, we note that both the VAERS database and the EU drug adverse events registry (EudraVigilance)

report fatalities in breastfed newborns shortly after vaccination of their mothers. Of course, these cases should have triggered a careful look for vaccine components in breast milk, and a targeted study of breast-fed infants of vaccinated mothers, but no such studies have been reported.



3.4 Summary

Pfizer's animal data clearly presaged the following risks and dangers:

- blood clotting shortly after vaccination, potentially leading to heart attacks, stroke, and venous thrombosis
- grave harm to female fertility
- grave harm to breastfed infants
- cumulative toxicity after multiple injections

With the exception of female fertility, which can simply not be evaluated within the short period of time for which the vaccines have been in use, and based on the very limited data available to the public, all of the above risks have been substantiated since the vaccines have been rolled out—all are manifest in the reports to the various adverse event registries [48].

We must emphasize that each of these risks could readily be inferred from the cited limited preclinical data, but were not followed up with appropriate in-depth investigations. In particular, the clinical trials did not monitor any laboratory parameters that could have provided information on these risks, such as those related to blood coagulation (e.g. D-dimers/thrombocytes), muscle cell damage (e.g. troponin/creatine kinase), or liver damage (e.g. γ-glutamyltransferase). The granting of emergency use authorization based on such incomplete and insufficient data by the various regulatory agencies amounts to nothing less than gross negligence.

Of particularly grave concern is the combination of cationic lipid accumulation in the ovaries with slow elimination and thus with cumulative toxicity. If girls and young women are repeatedly injected with mRNA vaccines containing these lipids—be they directed against COVID, or any other pathogen or disease—there is a real risk of massive damage to their fertility, or even of complete sterility. The plausible and grave risk to female fertility demands the most urgent attention of the public and of the health authorities.

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