# The Dangers of Covid-19 Booster Shots and Vaccines: Boosting Blood Clots and Leaky Vessels

## New discoveries in the immunology of SARS-CoV-2 and COVID-19 vaccines

1. Summary: Are COVID vaccines and booster shots safe and necessary? New discoveries in SARS-CoV-2 immunity and vaccine-immune interactions.
2. In Full: Explanation of new findings on the immunology of COVID-19 and its vaccines: How and why Covid-19 vaccines incite immunological attack on blood vessel walls. What is wrong with booster shots?
3. Implications for doctors and patients.

## 1. Summary: Are COVID Booster Shots and Vaccines Safe and Necessary? New Discoveries in SARS-CoV-2 Immunity and Vaccine-Immune Interactions

By now, most people have heard that COVID-19 vaccines can cause blood clotting and bleeding. Some readers may even be aware that reports of death following COVID-19 vaccination outnumber those for all vaccines combined since records began, 31 years ago, in the official US database VAERS [1,2].

With many patients now having received their first and second doses of COVID-19 vaccines, additional booster shots are being rolled out in many countries. Given that no clinical trials have been performed on more than two injections of any vaccine, it is important that doctors and patients understand how the vaccines interact with the immune system, and the implications for booster shots.

So far, doctors and patients confronted with information on COVID vaccine side effects are typically reassured that the benefits of COVID-19 vaccination outweigh the risks. Governments, the pharmaceutical industry, regulators and the media advise populations that the majority of adverse events are mild and transient, with serious complications in only a small minority of vaccine recipients.

Most patients, however, are unaware that among relevant scientific experts such a view is not so readily shared. Eminent independent scientists and researchers in the fields of immunology and microbiology have been writing to medical regulators since early 2021 [3], warning of vaccine-related blood clotting and bleeding, including that the official data on blood abnormalities post-vaccination likely represent “just the tip of a huge iceberg” [4]. Those scientists’ warnings pre-dated vaccine suspensions around the world due to acute disease from aberrant blood clotting post-vaccination. The warnings were based on established immunological science, applied to the novel mechanism of action of the gene-based COVID-19 vaccines.

Now, more than six months later, new discoveries in the immunology of SARS-CoV-2 [5] have caught up with the rushed vaccination schedule, confirming and extending the experts’ prior warnings. The good news is that we are more comprehensively protected against COVID-19 by our own pre-existing immunity than was previously understood. On the other hand, this pre-existing immunity aggravates the risk that COVID-19 vaccines will induce blood clotting and/or leaky blood vessels. This risk must be expected to escalate with each revaccination. Vaccine-induced harm to our blood vessels is unlikely to be rare.

Perhaps the most pertinent finding is that, due to the discovery of a widespread memory-type antibody response to SARS-CoV-2, the antibodies induced by the COVID-19 vaccines can be expected to activate the so-called complement system. This can bring about the destruction of any cell that manufactures the SARS-CoV-2 spike protein, particularly in the circulation. If that happens to the endothelia, that is, the cell layer that lines the inner surfaces of our blood vessels, then those vessels may begin to leak [6] and clots will form. Given that 2021 research showed the spike protein to enter the bloodstream shortly after vaccination [5], this dangerous endothelial involvement in spike-production is highly likely, and should be expected to occur.

As stark as these medical realities may be, the silver lining is that the same antibody profile, along with previously documented T-cell immunity [7–11], protects around 99% of the population against life-threatening SARS-CoV-2 infections. This ties in with the known fact that over 99% of people are safe from death with COVID-19 [12–14]. The implications for doctors and patients are that:

1. Vaccination against COVID-19 is unnecessary. Populations are protected by their immune systems against COVID-19. This applies to SARS-CoV-2 in all its variants.
2. Booster shots are uniquely dangerous, in a way that is unprecedented in the history of vaccines. This is because repeatedly boosting the immune response will repeatedly boost the intensity of self-to-self attack.

An important consideration for patients is that those who have already been vaccinated against COVID-19, and whose health remains intact, can protect themselves against serious harm by stopping now.

For a detailed explanation of the science behind these vaccine-immune interactions, please read Part II. Implications for doctors and patients are considered in Part III.

## 2. In Full: Explanation of New Findings on the Immunology of SARS-CoV-2 and COVID-19 Vaccines

### 2.1. How and why COVID-19 vaccines incite immunological attack on blood vessel walls. What is wrong with booster shots?

Until recently, the immune profile of COVID-19 and COVID-19 vaccines was not fully characterised. While we have known since mid-2020 that robust and lasting memory T-cell immunity to SARS-CoV-2 exists [7–11], the antibody picture has been less clear. Now, however, a convergence of evidence from peer reviewed studies published in 2021 reveals that pre-existing immunity to SARS-CoV-2 involves not only T-cells but also memory antibodies, in 99% of people studied. Two publications from 2020 alert to the probability that the immune response to the vaccine will also involve an important and powerful component called the complement system. This has profound consequences for the risk-benefit analysis of the vaccines.

Key papers behind these recent developments are:

1. Ogata et al. [15] showing that the SARS-CoV-2 spike protein circulates in the bloodstream shortly after vaccination with mRNA vaccine. This constitutes compelling evidence that spike protein molecules are produced by cells that are in contact with the bloodstream. The endothelial cells lining blood vessel walls naturally represent prime candidates.

2. Amanat et al. [16], Ogata et al. [15], and Wisnewski et al. [17], who found that circulating SARS-CoV-2-specific IgG and IgA antibodies became detectable within 1-2 weeks after mRNA vaccination. This early response indicates immunological memory—it can only be elicited through re-stimulation of pre-existing immune cells.
3. Gallais et al. [18], who provided data consistent with a memory-type antibody response in over 99% of people studied following first contact with the SARS-CoV-2 virus.
4. Wisnewski et al. [17], who reported a very rapid increase of spike protein antibodies after the second injection of mRNA vaccines. This finding underscores the immediate dangers of revaccination.
5. Magro et al. [19,20] showing that following entry into the bloodstream, spike protein directs complement attack to the inner vessel lining, causing damage and leakiness of the blood vessels

An explanation of the underlying immunology for laypeople follows.

## 2.2. Updated Immune Profile of COVID-19 and its Vaccines

Importantly for COVID-19 vaccination, the 2021 discoveries reveal that the SARS-CoV-2 virus responsible for COVID-19 is not truly new to our immune systems. The finding that the overwhelming majority of people show a memory-type antibody profile to COVID-19 vaccines proves that our immune systems have seen viruses similar to SARS-CoV-2 before. As a result, our bodies have stored an immune memory of that family of viruses, equipping us to fight back more rapidly and powerfully the next time we encounter a similar virus again. As SARS-Cov-2 is of the coronavirus family, this indicates that we possess lasting cross-immunity from previous exposure to other coronaviruses, such as common cold coronaviruses, which are in wide circulation globally. Simply put, almost anyone who is fundamentally healthy—or ‘immunocompetent’—is naturally sufficiently protected against COVID-19.

This immunological status accords with the well-documented reality that the infection fatality rate for COVID-19 is 0.15-0.2% worldwide [12–14]. As is well known, COVID-19 infection runs a fatal course only in those who are weakened by age and significant comorbidity. Put differently, once infected, COVID-19 is non-lethal to >99.8% of the world’s population. This same figure is upwards of 99.9% in the young and middle-aged. These statistics reflect the fact that protective cross-immunity is the global norm.

## 2.3. A Word on “Cases”

But what about the second and third waves of “cases”, including from Delta and other variants, around the world?

It is important to understand that a COVID-19 “case”, as currently defined, does not correspond to being ill. To an unprecedented extent in medical history, rather than referring to actual disease, the term “case” has become conflated with nothing more than a positive Polymerase Chain Reaction (PCR) test result. While PCR tests are useful in laboratory research and as diagnostic tools when carefully performed, they are not reliable or appropriate when used in isolation, nor set at extremely high sensitivities, nor in poorly trained hands, as has been the case overwhelmingly for COVID-19.

It has long been known that reliance on PCR tests alone to define medical “cases” and causes of death results in “overdiagnosis, overtreatment, and increased health care costs” [21]. If PCR alone were used to diagnose an infection with the diarrhoeal pathogen Clostridium difficile (CD), for instance, an epidemic of CD would immediately appear. We would find, based on PCR results, that 50% of people in long term care, and 15% of those hospitalised for any reason, are CD “cases” [22]. Should they die of any cause

following a positive PCR test for CD, they would be recorded as “dying with” CD. That figure could conceivably approach 100% if PCR tests were performed at the high sensitivities, or cycle thresholds, routinely employed when testing for COVID-19, in which the sensitivity of the test has been dialled up to meaningless extremes [23].

Moreover, even if we accepted PCR alone as a diagnostically appropriate tool—and therefore the high number of “cases” that it generates—we would still necessarily infer a very low infection fatality rate for COVID-19. This supports rather than contradicts the reality that SARS-CoV-2 poses no significant threat to the immunocompetent. In short, thanks to population immunity, for the vast majority of us, a “case” does not equate to severe disease.

## 2.4. Four Immunological Problems with COVID-19 Vaccines

While the now clearly established widespread cross-immunity against SARS-CoV-2 implies that most of us are safe from severe COVID-19 disease, it also means that we are vulnerable to the harms of gene-based vaccines. Due to recall immunity against the virus, vaccination will cause our immune systems to fight aggressively against not only the SARS-CoV-2 spike protein, but against ourselves. This deleterious autoimmune attack must be expected to intensify with each repeated injection.

The COVID-19 vaccine technology’s interaction with the immune system creates the following four specific problems:

1. Flying under the immune system’s radar with the vaccine’s genetic code
2. Delivering the spike protein into the bloodstream
3. Inducing immune attack on the blood vessel lining
4. Enhancing the severity of natural infection

### 2.4.1. Flying Under the Immune System’s Radar with the Vaccine’s Genetic Code

To understand why COVID-19 vaccine technology is dangerous, it is necessary to first understand how the gene-based vaccines differ from traditional vaccination methods.

A conventional viral vaccine can be a live virus strain derived from the pathogenic virus that has been *attenuated* through one or more genetic mutations, or it can consist of chemically inactivated virus particles that are no longer able to infect any cells. In both cases, protein antigens will be exposed on the surface of the vaccine particles, which can be recognized by antibodies once these have been formed.

COVID-19 vaccines, on the other hand, are not protein antigens but the genetic blueprint for the SARS-CoV-2 spike protein antigen. That blueprint comes in the form of mRNA or DNA, which, after vaccination, enters our body’s cells and instructs those cells to manufacture the spike protein. The spike protein then protrudes from the cell and induces antibody formation. In response, the immune system will react not only with the spike protein, but will attack and try to destroy the entire cell.

If we are injected with a traditional live virus vaccine to which we have no immunity, then these vaccine virus particles will also infect some of our body cells and propagate within them. Two kinds of immune reactions will then occur:

1. Cytotoxic T-lymphocytes (killer T-cells) (see section 2.4.3.1) that recognize viral protein fragments associated with the infected cells will proliferate, attack, and destroy the infected cells.
2. B-lymphocytes that recognize viral proteins (see section 2.4.3.2) will proliferate and start producing antibodies—soluble protein molecules that can recognize and neutralize virus particles.

This immune reaction will in principle resemble that to an infection with the corresponding wild-type virus. It will be milder, since the vaccine strain of the virus has been attenuated; however, some cells will get destroyed in the process, which may sometimes cause functional organ damage. Live virus vaccines therefore tend to be more prone to adverse reactions than are inactivated virus vaccines.

Now, a key point to note is that if we inject a live traditional vaccine into a person who is already immune—due to either a previous vaccination, or to prior infection with the corresponding wild-type virus—the extent of cell destruction will be much reduced. Such a person will already have antibodies to the virus; these will recognize the viral protein antigens and will bind and inactivate most of the vaccine virus particles before they manage to infect a cell. Therefore, even though the killer T-cells may be all riled up, they will not find very many infected cells to pounce on.

The crucial difference between a conventional live virus vaccine and a gene-based COVID vaccine—and in particular an mRNA vaccine—is that the latter contains no protein antigens whatsoever; instead, it only contains the blueprint for their synthesis inside the infected cells. Therefore, if such a vaccine is injected into a person with antibodies and existing T-cell immunity, the vaccine particles will "fly under the radar" of the antibody defence and reach our body cells unimpeded. The cells will then produce the spike protein, and subsequently be destroyed and attacked by the killer T-cells. The antibodies, rather than preventing the carnage, will join in by also binding to the cell-associated spike protein and directing the complement system (see later) and other immune effector mechanisms against these cells. In a nutshell, pre-existing immunity mitigates the risk of conventional vaccines, but it amplifies the risk of gene-based vaccines.

Importantly, before COVID, this risky gene-based vaccine technology had never before been used on a wide scale against infectious disease and is inherently experimental. The COVID-19 vaccination program is thus the largest human experiment ever performed in history.

### 2.4.2. Delivering the Spike Protein into the Bloodstream

A dire danger of COVID-19 vaccines is that spike proteins produced by myriad endothelial cells, i.e. the innermost cells lining blood vessel walls, will be exported to the cell surface and protrude directly into the bloodstream. Moreover, a fraction of these spikes will be cleaved during their passage to the outside world. They will fall off the cells into the bloodstream and then bind to their receptors on other endothelial cells at distant sites.

While at the outset of the vaccination campaign in 2020 it was unknown to what extent COVID vaccines entered the bloodstream, human data from 2021 reveal that the spike protein shows up within the circulation on the very day of the injection [15]. Similarly, animal studies submitted by Pfizer to the Japanese government [24] found that the vaccine appears in the circulation within 15 minutes of intramuscular injection, reaching maximum plasma concentration within just two hours. Very high levels have subsequently been recorded in the liver, the spleen, the adrenal glands, and the ovaries. Vaccine components have also been observed in the central nervous system (the brain and the spinal cord), albeit at lower concentrations. Such widespread distribution throughout the body via the bloodstream is a feat that the SARS-CoV-2 virus does not usually achieve.

#### 2.4.2.1. Open Questions in the Ongoing Experiment

But how do COVID-19 vaccine particles enter the circulation in the first place? The vaccine is injected intramuscularly, and the vaccine particles are too large to passively diffuse across blood vessel walls. Most obviously, the vaccines will follow the conventional, relatively time-consuming path which takes them via the draining lymph nodes to the blood circulation. But additionally, two possibilities for very rapid entry

into the bloodstream should be heeded. The first is via direct uptake by vessels that are damaged during insertion of the needle. Secondly, it is possible that the vaccine particles undergo ‘transcytosis’, a process that enables large molecules to be transported across intact cell layers. Whatever the case may be, although Pfizer knew before the onset of clinical trials that their vaccine reached the bloodstream rapidly, either they failed to file these findings with medical regulators in Europe, the US and other Western countries, or the regulators failed to act upon the findings [25].

This is a critical oversight where patient safety is concerned. Given that the gene-based vaccines induce the body’s cells to become immune targets, where in the body this takes place is of critical concern. While immune-mediated cell death is never favourable, it is particularly detrimental and dangerous if it afflicts the blood vessel walls.

### 2.4.3. Attacking the Vessel Walls: Clotting and Leaky Vessels

While all vaccines seek to stimulate an immune response, not all immune responses are created equal. Some are safe and well-modulated whereas others can be misdirected and out of control. Immune responses are problematic when they attack the self, as in autoimmune conditions, and/or when they are excessively intense and severe.

COVID-19 vaccines incur problematic immunity in both key ways. First, they can be expected mobilise a self-to-self immune response against the endothelial cells lining blood vessel walls. Second, by boosting SARS-CoV-2 immunity, they can be expected to incite an increasingly aggressive response with each administration of the vaccine.

To understand the realities of these processes it is necessary to first understand the basics of the underlying immune response. There are three key components of the immune system relevant to risks from COVID-19 vaccines: T-cells, antibodies and the complement cascade.

#### 2.4.3.1. T-cells

Once the body’s cells have been infected with a virus, immune cells known as cytotoxic T-cells or T-killer cells attack and destroy the infected cells. This prevents infected cells from replicating the virus and spreading the infection throughout the body. After the initial battle with a certain pathogen is over, some of the specifically adapted T-cells enter a state of dormancy to become memory T-cells. In case the same virus is encountered again, these dormant T-cells can be swiftly reawakened and propagated to mount a faster and more vigorous response next time. Known as a secondary or memory-type response, it will also occur with viruses that are not exactly the same as the one initially encountered but sufficiently similar to be recognised. This latter phenomenon is referred to as cross-immunity.

It has been known since mid 2020 that we are protected against SARS-CoV-2 by cross-reactive memory T-cells [7–11]. As with antibodies, this is based on previous encounters with common cold coronaviruses, and with the SARS virus in a small number of people. Such prior experience has been found to confer “robust” [7] and lasting T-cell cross-immunity to COVID-19. T-cell memory for the SARS virus is known to last at least 17 years [7], but it likely lasts a lifetime.

#### 2.4.3.2. Antibodies

Before the new discoveries of 2021, scientists’ concerns about clotting and bleeding were based primarily on the prediction that killer T-cells would attack spike-producing endothelial cells, causing lesions on

vessel linings and promoting blood clots. While this mechanism remains valid, we now know that a memory-type antibody response will join the attack on the vessel walls as well.

Whereas killer T-cells attack their targets cell-to-cell, antibodies are proteins that exert their effect by binding to signature structures on the pathogen's surface, known as epitopes. Instead of destroying cells directly, once attached to an epitope, antibodies help to defeat invaders by "calling out the cavalry" on infected cells.

This leads to the second process by which cells coated with viral spikes will inadvertently come under immune attack. "Calling out the cavalry" means that the antibodies attached to the unnaturally created spikes will trigger activation of the complement system, which thereupon will mount a massive attack on the endothelial cells.

Importantly for deciphering the recent discoveries on SARS-CoV-2 immunity, the first time that the immune system encounters a new pathogen, new antibodies in a shape capable of binding to that pathogen's epitopes must be formed (by immune cells known as B-cells). First-time antibody production is slow, taking approximately four weeks. Should the same pathogen or family of pathogens invade again, however, memory-type antibodies are then manufactured more rapidly, within one to two weeks. This is a cardinal sign that the immune system has seen that pathogen before.

Another defining feature of a memory antibody response concerns the order in which antibody sub-types are produced. If a pathogen is new, IgM is the first type of antibody to arrive on the scene. It is followed later by IgG and IgA. The next time the pathogen arrives, however, IgG and IgA will be the first to arrive, indicating that the virus, or its relatives, have invaded before.

Importantly, this is precisely what we see with COVID-19.

Several research groups found in 2021 that upon first exposure to SARS-CoV-2, and following COVID-19 vaccination, the antibody response was characteristic of the memory type, due both to the timing and nature of antibodies measured. [xv-xvii] As a result, we now know that our immune systems recognise SARS-CoV-2 at first sight, even "on the slightest viral challenge" [5]. In other words, SARS-CoV-2 is not a novel coronavirus after all.

With respect to variants and the need for booster shots, memory B-cells, like memory T-cells, can recognise not only a specific virus, but a whole family of viruses bearing related epitopes. It is unsurprising, therefore, that memory B-cells recognise SARS-CoV-2 from the common cold. With cross immunity this robust, closer relatives of SARS-CoV-2 in the form of variants will pose no obstacle to our antibody response. The rising "cases", hospitalisations and deaths attributed to Delta and other variants are therefore almost certainly driven by false positive PCR results and misclassification than by a true increase in COVID-19 disease. Indeed, according to Public Health England data, the Delta variant is non-lethal in those under 50, and less than half as lethal as earlier strains in older age groups [26].

But why haven't circulating antibodies to SARS-CoV-2 been detected in populations before? The answer is that neither the antibodies nor T-cells associated with a memory-type response circulate in the bloodstream. Once they are no longer needed, they become dormant, existing as a memory alone. Unless elicited by re-exposure to a virus, they remain invisible in the bloodstream. The dormant antibodies will, however, be ready and waiting to re-activate and call out the cavalry on the spike protein, in the form of the complement cascade.

#### 2.4.3.3. Complement

Recent findings indicate that complement activation is a serious concern with respect to COVID-19 vaccine-immune interactions.

In light of the newly characterised antibody response to SARS-CoV-2, when antibodies attach to spike-producing endothelial cells on vessel walls following vaccine administration, activated complement proteins can be expected attach to the endothelial cells, and perforate their cell membranes [27,28]. The ensuing death of the endothelial cells will expose the tissue underneath the epithelium, which will initiate two significant events. It will induce blood clotting, and will cause the vessel walls to leak [6]. This pathogenic mechanism has been documented in biopsies taken from SARS-CoV-2-infected patients [19,29]. Those studies have described a "catastrophic microvascular injury syndrome mediated by activation of complement" [29] as part of the SARS-CoV-2 spike protein immune response. It is precisely this immune response that COVID-19 vaccines seek to induce.

Such vaccine-immune interactions are consistent with adverse events involving visible capillary rupture under the skin that have been documented and reported following COVID-19 vaccination [30–33].

#### 2.4.3.4. Leaky Vessels—The Promise of Booster Shots

Given that booster shots repeatedly boost the immune response to the spike protein, they will progressively boost self-to-self immune attack, including boosting complement-mediated damage to vessel walls.

Clinically speaking, the greater the vessel leakage and clotting that subsequently occurs, the more likely that organs supplied by the affected blood flow will sustain damage. From stroke to heart attack to brain vein thrombosis, the symptoms can range from death to headaches, nausea and vomiting, all of which heavily populate adverse reactions to COVID-19 vaccines [2].

As well as damage from leakage and clotting alone, it is additionally possible that the vaccine itself may leak into surrounding organs and tissues. Should this take place, the cells of those organs will themselves begin to produce spike protein, and will come under attack in the same way as the vessel walls. Damage to major organs such as the lungs, ovaries, placenta and heart can be expected ensue, with increasing severity and frequency as booster shots are rolled out.

### 2.4.4. Enhancing the Severity of Wild Coronavirus Infection

Finally, as with the Dengue virus and several other viruses [34], antibodies to coronaviruses can ultimately aggravate rather than mitigate illness. This is called antibody-dependent enhancement of disease. The underlying mechanisms remain to be elucidated but it is already clear that the net effects are severely detrimental.

Attempts to develop vaccines to the original SARS virus, which is closely related to SARS-CoV-2, repeatedly failed due to antibody-dependent enhancement of disease [35–37]. The vaccines induced antibodies, but when the vaccinated animals were subsequently infected with the wild-type virus, they became more ill than the unvaccinated animals, in some cases mortally so [38].

## 3. Implications for Doctors and Patients

Although vaccine manufacturers and regulators are aware of the risks of antibody enhancement of disease, this possibility was not adequately addressed in the clinical trials on any of the COVID-19 vaccines. The

FDA noted that Pfizer, “identified vaccine-associated enhanced disease, including vaccine-associated enhanced respiratory disease, as an important potential risk” [23]. The EMA similarly acknowledged that “vaccine associated enhanced respiratory disease” was “an important potential risk… that may be specific to vaccination for COVID- 19”.

Why neither regulator sought to exclude such dangers prior to emergency use authorisation is an open question that all doctors and patients are entitled to ask. Why medical regulators failed to investigate the finding that large vaccine particles cross blood vessel walls, entering the bloodstream and posing risks of blood clotting and leaky vessels is yet another open question again.

The fact that vaccine rollout began before the immune profile of SARS-CoV-2 and COVID-19 vaccines had been adequately delineated is symptomatic of a rushed and highly politicised approach to the approval and regulation of COVID-19 vaccines. As is the lack of clinical trials investigating the safety of COVID-19 booster shots.

In this context, it is up to doctors and patients to uphold the social contract of the doctor-patient relationship, and take medical prudence and patient safety into their own hands.

The World Medical Association, Declaration of Geneva, Physician’s Pledge states [39]:

“The health and wellbeing of my patient will be my first consideration. I will maintain the utmost respect for human life. I will practise my profession with conscience and dignity and in accordance with good medical practice. I will respect the autonomy and dignity of my patient. I will not use my medical knowledge to violate human rights and civil liberties, even under threat.”

## References


1. Open VAERS, (2021) VAERS COVID vaccine data.
2. Open VAERS, (2021) All deaths reported to VAERS by year
3. Doctors for Covid Ethics, (2021) Doctors for COVID Ethics: letters.
4. Doctors for Covid Ethics, (2021) Rebuttal letter to European Medicines Agency from Doctors for Covid Ethics, April 1, 2021.
5. Bhakdi, S. et al. (2021) Letter to Physicians: Four New Scientific Discoveries Regarding COVID-19 Immunity and Vaccines—Implications for Safety and Efficacy.
6. Doctors for Covid Ethics, (2021) Leaky Blood Vessels: An Unknown Danger of COVID-19 Vaccination.
7. Le Bert, N. et al. (2020) SARS-CoV-2-specific T cell immunity in cases of COVID-19 and SARS, and uninfected controls. *Nature* 584:457-462
8. Tarke, A. et al. (2021) Impact of SARS-CoV-2 variants on the total CD4+ and CD8+ T cell reactivity in infected or vaccinated individuals. *Cell reports. Medicine* 2:100355
9. Grifoni, A. et al. (2020) Targets of T Cell Responses to SARS-CoV-2 Coronavirus in Humans with COVID-19 Disease and Unexposed Individuals. *Cell* 181:1489-1501.e15
10. Mateus, J. et al. (2020) Selective and cross-reactive SARS-CoV-2 T cell epitopes in unexposed humans. *Science* 370:89-94
11. Sekine, T. et al. (2020) Robust T Cell Immunity in Convalescent Individuals with Asymptomatic or Mild COVID-19. *Cell* 183:158-168.e14
12. Ioannidis, J.P.A. (2020) Infection fatality rate of COVID-19 inferred from seroprevalence data. *Bull. World Health Organ.* -:BLT.20.265892

13. Ioannidis, J.P.A. (2020) Global perspective of COVID-19 epidemiology for a full-cycle pandemic. *Eur. J. Clin. Invest.* 50:x-x
14. Ioannidis, J.P.A. (2021) Reconciling estimates of global spread and infection fatality rates of COVID-19: An overview of systematic evaluations. *Eur. J. Clin. Invest.* 5:e133554
15. Ogata, A.F. et al. (2021) Circulating SARS-CoV-2 Vaccine Antigen Detected in the Plasma of mRNA-1273 Vaccine Recipients. *Clin. Infect. Dis.* -:x-x
16. Amanat, F. et al. (2021) SARS-CoV-2 mRNA vaccination induces functionally diverse antibodies to NTD, RBD and S2. *Cell* -:x-x
17. Wisnewski, A.V. et al. (2021) Human IgG and IgA responses to COVID-19 mRNA vaccines. *PLoS One* 16:e0249499
18. Gallais, F. et al. (2021) Intrafamilial Exposure to SARS-CoV-2 Associated with Cellular Immune Response without Seroconversion. *Emerg. Infect. Dis.* 27:x-x
19. Magro, C.M. et al. (2020) Docked severe acute respiratory syndrome coronavirus 2 proteins within the cutaneous and subcutaneous microvasculature and their role in the pathogenesis of severe coronavirus disease 2019. *Hum. Pathol.* 106:106-116
20. Magro, C.M. et al. (2021) Severe COVID-19: A multifaceted viral vasculopathy syndrome. *Annals of diagnostic pathology* 50:151645
21. Polage, C.R. et al. (2015) Overdiagnosis of Clostridium difficile Infection in the Molecular Test Era. *JAMA internal medicine* 175:1792-801
22. Anonymous, (2021) Overdiagnosis of Clostridium difficile.
23. Palmer, M. et al. (2021) Expert evidence regarding Comirnaty (Pfizer) COVID-19 mRNA Vaccine for children.
24. Anonymous, (2020) SARS-CoV-2 mRNA Vaccine (BNT162, PF-07302048) 2.6.4 Summary statement of the pharmacokinetic study [English translation].
25. Palmer, M. and Bhakdi, S. (2021) The Pfizer mRNA vaccine: Pharmacokinetics and Toxicity.
26. Public Health England, (2021) SARS-CoV-2 variants of concern and variants under investigation in England.
27. Bhakdi, S. and Tranum-Jensen, J. (1978) Molecular nature of the complement lesion. *Proc. Natl. Acad. Sci. U. S. A.* 75:5655-5659
28. Tranum-Jensen, J. et al. (1978) Complement lysis: the ultrastructure and orientation of the C5b-9 complex on target sheep erythrocyte membranes. *Scandinavian journal of immunology* 7:45-6
29. Magro, C. et al. (2020) Complement associated microvascular injury and thrombosis in the pathogenesis of severe COVID-19 infection: A report of five cases. *Transl Res* 220:1-13
30. Greinacher, A. et al. (2021) Thrombotic Thrombocytopenia after ChAdOx1 nCov-19 Vaccination. *N. Engl. J. Med.* -:x-x
31. Lee, E. et al. (2021) Thrombocytopenia following Pfizer and Moderna SARS-CoV-2 vaccination. *Am. J. Hematol.* -:x-x
32. Malayala, S.V. et al. (2021) Purpuric Rash and Thrombocytopenia After the mRNA-1273 (Moderna) COVID-19 Vaccine. *Cureus* 13:e14099
33. Tarawneh, O. and Tarawneh, H. (2021) Immune thrombocytopenia in a 22-year-old post Covid-19 vaccine. *Am. J. Hematol.* 96:E133-E134
34. Tirado, S.M.C. and Yoon, K. (2003) Antibody-dependent enhancement of virus infection and disease. *Viral immunology* 16:69-86
35. Tseng, C. et al. (2012) Immunization with SARS coronavirus vaccines leads to pulmonary immunopathology on challenge with the SARS virus. *PLoS One* 7:e35421

36. Weingartl, H. et al. (2004) Immunization with modified vaccinia virus Ankara-based recombinant vaccine against severe acute respiratory syndrome is associated with enhanced hepatitis in ferrets. *J. Virol.* 78:12672-6
37. Czub, M. et al. (2005) Evaluation of modified vaccinia virus Ankara based recombinant SARS vaccine in ferrets. *Vaccine* 23:2273-9
38. Bolles, M. et al. (2011) A double-inactivated severe acute respiratory syndrome coronavirus vaccine provides incomplete protection in mice and induces increased eosinophilic proinflammatory pulmonary response upon challenge. *J. Virol.* 85:12201-15
39. World Medical Association, (2017) WMA Declaration of Geneva.