Are The Vaccines Safe and Effective?

Questions To Answer

1. **Why is there concern surrounding this particular vaccine?**
2. **Do the vaccines cause blood clots?**
3. **Are vaccinated individuals shedding spike protein?**
4. **Is the spike protein toxic?**
5. **What are the effects of the vaccines on your immune system?**
6. **What do the autopsies show?**
7. **Can the mRNA be converted into DNA?**

Why is there concern surrounding this particular vaccine?

The COVID-19 vaccines are still experimental. **The injections are still in Stage 3 Clinical Trials that do not finish until late 2022 to mid 2023.**
Pfizer - https://clinicaltrials.gov/ct2/show/NCT04368728 (Ends May 2nd, 2023)
Moderna - https://clinicaltrials.gov/ct2/show/NCT04470427?term=moderna&draw=4&rank=102 (Ends Oct. 27, 2022)

The public should only begin to use drugs or vaccines that are in Stage 4 clinical trials

Pfizer tells us this : Stage 3 not normal. https://www.pfizer.co.uk/clinical-trials

Phase 3: 1,000 - 5,000 patients[4]
Phase 3 trials test the results of earlier trials in much larger groups of people and gather additional information on the effectiveness and safety of the experimental therapy.

This phase will usually involve several hundred to several thousand participants across multiple study locations. These trials are often randomized, where participants are randomly allocated to receive the experimental therapy, placebo, or an existing treatment. Trials are often "double-blinded" in which neither the investigator nor the participant are aware if the therapy given is the true experimental therapy, placebo, or an existing therapy.[5] Phase 3 trials generally provide the primary basis for the benefit–risk assessment for the new therapy and much of the core information about the therapy that is analysed for inclusion in final labeling if approved by the regulatory authority.

The animals trials for coronavirus vaccines have always failed because the animals got sick and/or died

Studies in Ferrets : https://journals.asm.org/doi/full/10.1128/JVI.78.22.12672-12676.2004
Ferrets vaccinated with rMVA-S and exposed to SARS-CoV had elevated levels of an enzyme that indicates liver damage. Examination of liver sections showed that the ferrets had severe hepatitis. Only mild hepatitis was found in the ferrets injected with parental MVA or saline.

Cats: https://www.jstage.jst.go.jp/article/jvms/60/1/60_1_49/_article
These findings support the previous results where a monoclonal antibody with neutralizing activity had high ADE (Antibody Dependent Enhancement) activity, suggesting that there was a close relationship between the neutralization and enhancement sites. And then it is also suggested that ADE of infection is likely to be induced by re-infection with the same serotype of virus in type II FIPV infection.

Understanding Antibody Dependent Enhancement (ADE):
https://www.nature.com/articles/s41564-020-00789-5
https://www.youtube.com/watch?v=qOLksb6PMoA

The animals trials for coronavirus vaccines have always failed because the animals got sick and/or died

Mice : https://pubmed.ncbi.nlm.nih.gov/22536382/
These SARS-CoV vaccines all induced antibody and protection against infection with SARS-CoV. **However, challenge of mice given any of the vaccines led to occurrence of Th2-type immunopathology suggesting hypersensitivity to SARS-CoV components was induced.** Caution in proceeding to application of a SARS-CoV vaccine in humans is indicated.

Mice: https://pubmed.ncbi.nlm.nih.gov/17194199/
VRP-N vaccines not only failed to protect from homologous or heterologous challenge, but resulted in enhanced immunopathology with eosinophilic infiltrates within the lungs of SARS-CoV-challenged mice.

Mice: https://pubmed.ncbi.nlm.nih.gov/18941225/
Furthermore, LC16mOrVV-N-**immunized mice upon infection exhibited significant up-regulation of both Th1 (IFN-gamma, IL-2) and Th2 (IL-4, IL-5) cytokines and down-regulation of anti-inflammatory cytokines (IL-10, TGF-beta), resulting in robust infiltration of neutrophils, eosinophils, and lymphocytes into the lung, as well as thickening of the alveolar epithelium.** These results suggest that an excessive host immune response against the nucleocapsid protein of SARS-CoV is involved in severe pneumonia caused by SARS-CoV infection.

The trials for Comirnaty were short and flawed

DOSES & DURATION OF IMMUNITY

Two doses are required.

The **duration of immunity is unknown**, due to limited data, as the studies have not yet been completed. Immunity is believed to last at least 2 months.

ANIMAL TRIALS

No animal safety studies have been published for this vaccine. The vaccine was tested on **mice and macaque monkeys to establish efficacy** only (not safety)[iii].

Trials on mRNA vaccines for other coronaviruses resulted in severe side-effects, due to a phenomenon called **Antibody Dependent Enhanced Immunity**[iv]. This occurs when the vaccine-induced antibodies paradoxically cause a **more severe illness with subsequent exposure to the wild virus**. Many animals involved died or became very unwell and human trials did not proceed.

Pfizer reported (see CDC presentation[v]) a **brief safety study on Wistar rats** with no systemic events identified, however **this study is not publicly available**.

Efficacy studies: both mice and macaques developed antibodies to SARS-CoV-2 following immunisation. After vaccination, when deliberately exposed to SARS-CoV-2, all the macaque monkeys had evidence of infection with SARS-CoV-2 in their noses and airways. **None of the monkeys, in either the vaccinated or placebo group, developed symptoms.** The animals that received the **vaccine had evidence of SARS-CoV-2 in their noses for 1 day,** while those that received the **placebo had SARS-CoV-2 on nasal swabs on days 1, 3 and 6 after exposure**. As no animals in either group developed symptoms, we cannot infer whether the vaccine would reduce symptoms in humans. **No assessment of transmission was performed.**

The trials for Comirnaty were short and flawed

HUMAN TRIALS

Phase 1 & 2 Trials:

Across the Phase 1 and 2 trials[vi], **204 people received 2 doses of the BNT162b2 vaccine** (others received a placebo or a potential alternative vaccine). Participants provided a daily diary, for a list of side-effects, over 7 days[vii] after receiving the vaccine. Some reported **headaches, muscle and joint pain** which was **debilitating and prevented them from performing even basic daily chores**. Phase 2 **efficacy will be measured for 24 months**, while **safety will be monitored for 6 months**.

Lymphocyte numbers were monitored in the 24 people who received the vaccine in Phase 1**, otherwise no blood tests to assess the potential impact on internal organs or other aspects of health are being conducted** at any point.

Phase 3:

43,538 people had been enrolled in the Phase 3 trial by early December 2020[viii]. **The study does not end until January 2023**; people who take the vaccine should be aware that it is still essentially experimental.

Efficacy:

Pfizer published a **press release stating the vaccine is 95% effective. This efficacy calculation is based on only 181 of the 43,548 participants** (see below).

Of 43,548 trial participants, only **181 people had confirmed cases of symptomatic SARS-CoV-2 infection (COVID-19)** - 1 or more symptoms plus a positive PCR result at least 7 days after the second vaccine dose. Of the 181 "cases", 95% were in the placebo group and 5% in the vaccine group. Several other participants developed COVID-19, but were **excluded from the analysis** due to timing of their symptoms.

The following groups were excluded from the trials as such **no data exists for the safety and efficacy of the vaccine in these groups**:

- **Anyone considered a suicide risk or with mental health disorders**
- **Pregnant and breastfeeding women**
- **Children under the age of 12**
- **Anyone with evidence of a prior COVID infection**

The Pfizer booster shot clinical trial only included "Healthy participants who are determined by medical history, physical examination (if required), and clinical judgment of the investigator to be eligible for inclusion in the study."

Data from the clinical trial was limited in size (~300) and age (primarily 18-55 years) -
https://www.cdc.gov/vaccines/acip/meetings/downloads/slides-2021-9-23/03-COVID-Oliver.pdf
It excluded:

- Other medical or psychiatric conditions including recent (within the past year) or active suicidal ideation/behavior or laboratory abnormality that may increase the risk of study participation or, in the investigator's judgment, make the participant inappropriate for the study.
- Phases 1 and 2 only: Known infection with human immunodeficiency virus (HIV), hepatitis C virus (HCV), or hepatitis B virus (HBV).
- History of severe adverse reaction associated with a vaccine and/or severe allergic reaction (eg, anaphylaxis) to any component of the study intervention(s).
- Receipt of medications intended to prevent COVID 19. (**What medications are these?)**
- **Previous clinical or microbiological diagnosis of COVID 19**
- Phase 1 only: Individuals at high risk for severe COVID-19, including those with any of the following risk factors:
    - **Hypertension**
    - **Diabetes mellitus**
    - **Chronic pulmonary disease**
    - **Asthma**
    - **Current vaping or smoking**
    - **History of chronic smoking within the prior year**
    - **BMI >30 kg/m2**
    - **Anticipating the need for immunosuppressive treatment within the next 6 months**

- **Phase 1 only: Individuals currently working in occupations with high risk of exposure to SARS-CoV-2 (eg, healthcare worker, emergency response personnel).**
- **Immunocompromised individuals with known or suspected immunodeficiency, as determined by history and/or laboratory/physical examination.**
- **Phase 1 only: Individuals with a history of autoimmune disease or an active autoimmune disease requiring therapeutic intervention.**
- Bleeding diathesis or condition associated with prolonged bleeding that would, in the opinion of the investigator, contraindicate intramuscular injection.
- **Women who are pregnant or breastfeeding.**
- **Previous vaccination with any coronavirus vaccine.**
- I**ndividuals who receive treatment with immunosuppressive therapy, including cytotoxic agents or systemic corticosteroids, eg, for cancer or an autoimmune disease, or planned receipt throughout the study.**
- **Phase 1 only: Regular receipt of inhaled/nebulized corticosteroids.**
- **Receipt of blood/plasma products or immunoglobulin, from 60 days before study intervention administration or planned receipt throughout the study.**
- **Previous participation in other studies involving study intervention containing lipid nanoparticles.**
- **Phase 1 only: Positive serological test for SARS-CoV-2 IgM and/or IgG antibodies at the screening visit.**

Pfizer Booster Shot

What is concerning is that many of the people excluded from the booster clinical trial are precisely the individuals that they recommend to get the booster.

> **“CDC recommends that the following groups should receive a booster dose of Pfizer-BioNTech’s COVID-19 vaccine at least 6 months after completing their Pfizer-BioNTech primary vaccine series:**

- People aged 65 years and older
- Residents aged 18 years and older in long-term care settings
- People aged 50–64 years with underlying medical conditions”

https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-care/underlyingconditions.html

Do Vaccines Cause Blood Clots?

Antibody epitopes in vaccine-induced immune thrombotic thrombocytopenia

Vaccine-induced immune thrombotic thrombocytopaenia (VITT) is a rare adverse effect of COVID-19 adenoviral vector vaccines 1,2,3. **Our data indicate that VITT antibodies can mimic the effect of heparin by binding to a similar site on PF4; this allows PF4 tetramers to cluster and form immune complexes, which in turn causes Fcγ receptor IIa (FcγRIIa; also known as CD32a)-dependent platelet activation.** These results provide an explanation for VITT-antibody-induced platelet activation that could contribute to thrombosis. The time from first dose of the ChAdOx1 nCoV-19 **vaccine to sample collection was 14–40 days** (mean, 28 days). All samples from patients with VITT (hereafter, VITT samples) had antibodies against PF4.

https://www.nature.com/articles/s41586-021-03744-4#Sec1

Do Vaccines Cause Blood Clots?

Recognizing Vaccine-Induced Immune Thrombotic Thrombocytopenia

Vaccine-induced immune thrombotic thrombocytopenia is a serious complication of vaccination that is not feasible to anticipate or prevent. When the patient presents with sustained headache, neurologic symptoms/signs, abdominal pain, dyspnea, or limb pain/swelling beginning 5-30 days post vaccination, platelet count and D-dimer must be measured, and imaging for thrombosis performed.

https://pubmed.ncbi.nlm.nih.gov/34259661/

Do Vaccines Cause Blood Clots?

A COVID-Positive 52-Year-Old Man Presented With Venous Thromboembolism and Disseminated Intravascular Coagulation Following Johnson & Johnson Vaccination: A Case-Study

The use of the vaccine was halted after reported cases of cerebral venous sinus thrombosis (CVST) and thrombocytopenia among recipients. Researchers have postulated these rare occurrences as potentially immune-triggered responses associated with complement-mediated thrombotic microangiopathy (TMA). **Thrombotic complications and thrombocytopenia increase the risk for blood clot growth due to the inflammation of immune complexes by pro-thrombotic activation of anti-platelet antibodies.** A 52-year-old man presented to the intensive care unit (ICU) with severe dyspnea. He required bilevel positive airway pressure (BiPAP) for supplemental oxygen therapy. Endotracheal intubation was performed due to his worsened respiratory deterioration. Lab results suggested respiratory failure due to decreased partial pressure of oxygen (pO_2) and increased partial pressure of carbon dioxide (pCO_2). **Findings of elevated D-dimer levels with decreased fibrinogen and thrombocytopenia with prolonged prothrombin clotting time were consistent for disseminated intravascular coagulation (DIC).**
https://pubmed.ncbi.nlm.nih.gov/34408937/

Do Vaccines Cause Blood Clots?

The roles of platelets in COVID-19-associated coagulopathy and vaccine-induced immune thrombotic thrombocytopenia

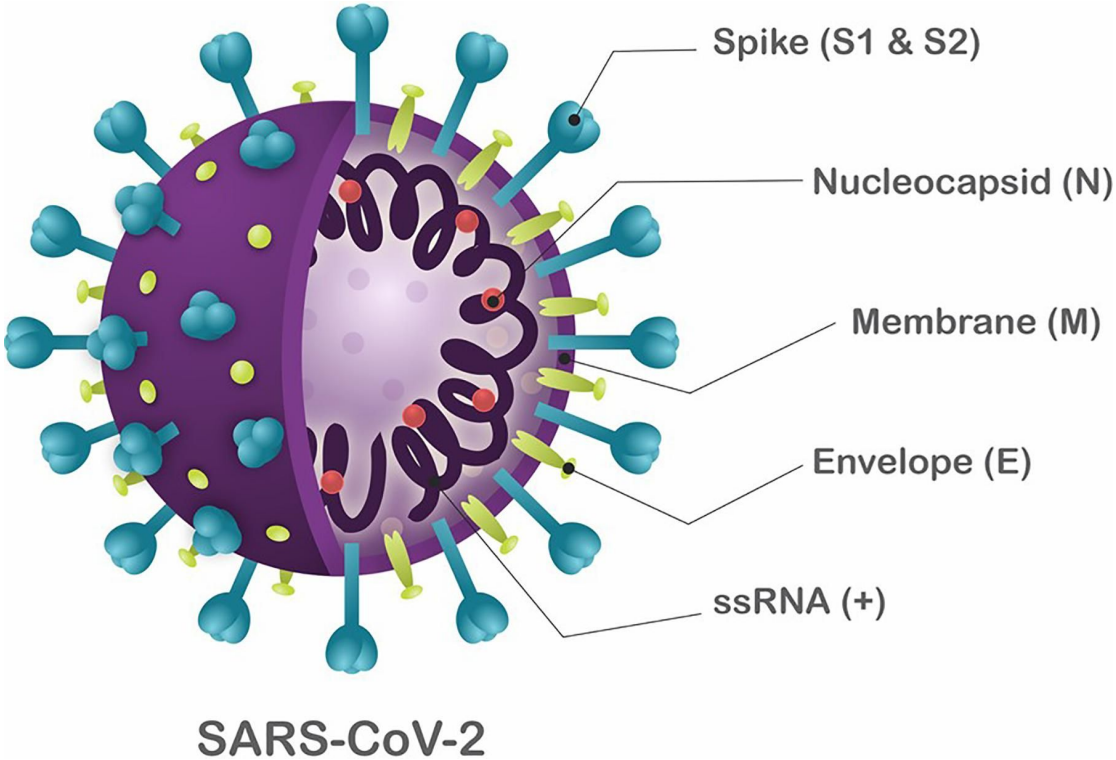
Although clinical features of vaccine-induced immune thrombotic thrombocytopenia include uncommon locations of thrombosis, including cerebral venous sinus, **we speculate coronavirus spike-protein-initiated prothrombotic pathways are involved in the pathogenesis of vaccine-induced immune thrombotic thrombocytopenia, as current evidence suggests that the spike protein is the promotor and other cofactors such as perturbed immune response and inflammatory reaction enhance the production of anti-platelet factor 4 antibody.**

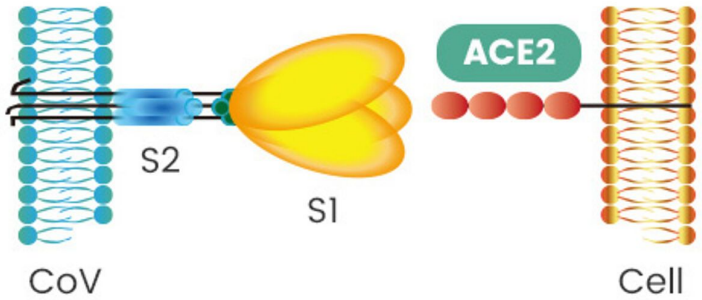
As described previously, **the spike protein of coronavirus upregulates the inflammatory response and injures the vascular endothelium by binding to ACE2** [54]. ACE2 converts angiotensin II to angiotensin 1–7, a molecule counterbalancing the vasoconstrictive, proinflammatory, and pro-coagulant effects of angiotensin II [55]. Therefore, decline of angiotensin 1–7 on the cellular membrane by COVID-19 infection, as well as loss of the endothelial anticoagulant effects, leads to microcirculatory disturbance by vasoconstriction, profound inflammation, and activated coagulation [56]. The vaccine-induced spike protein also may induce the similar reactions and may serve the underlying condition of thrombogenesis. Colunga Biancatelli et al. [57] reported the S1 subunit of SARS-CoV-2 spike protein alone could produce acute lung injury. **https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8390120/**

Do Vaccines Cause Blood Clots?

Thrombosis With Thrombocytopenia After the Messenger RNA–1273 Vaccine

He developed thrombocytopenia and thrombosis within 5 to 10 days after vaccine administration. **The distribution of thrombosis, especially the cerebral venous sinus thrombosis, was characteristic of VITT or TTS.** Most of his clotting and other relevant work-up were consistent with the syndrome. **We were unable to identify other causes, including SARS-CoV-2 infection**, other infections, immune thrombocytopenia, or thrombotic thrombocytopenic purpura. These findings fulfill the interim case definition of VITT or TTS from the Centers for Disease Control and Prevention and the Brighton Collaboration. **Further, the positive platelet factor 4 enzyme-linked immunosorbent assay of the blood drawn before heparin administration strengthens the likelihood of VITT or TTS.**
https://www.acpjournals.org/doi/10.7326/L21-0244


Spike (S1 & S2)
Nucleocapsid (N)
Membrane (M)
Envelope (E)
ssRNA (+)
SARS-CoV-2


ACE2
S2
S1
CoV
Cell

Are Vaccinated Individuals Shedding Spike Protein

Study: Fully Vaccinated Healthcare Workers Carry 251 Times Viral Load, Pose Threat to Unvaccinated Patients, Co-Workers

Viral loads of breakthrough Delta variant infection cases were 251 times higher than those of cases infected with old strains detected between March-April 2020. **Neutralizing antibody levels after vaccination and at diagnosis of the cases were lower than those in the matched uninfected controls.**

Breakthrough Delta variant infections are associated with high viral loads, prolonged PCR positivity, and low levels of vaccine-induced neutralizing antibodies, explaining the transmission between the vaccinated people.

https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3897733

Are Vaccinated Individuals Shedding Spike Protein

Breakthrough Infections in BNT162b2-Vaccinated Health Care Workers

The health care workers at our institution had only mild symptoms **but high viral loads (cycle thresholds of <25) and prolonged viral shedding up to 32 days after diagnosis.**

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8385562/

Are Vaccinated Individuals Shedding Spike Protein

Acute kidney injury with gross hematuria and IgA nephropathy after COVID-19 vaccination

The mRNA coronavirus disease 2019 (COVID-19) vaccines induce an IgG response that prevents people from contracting severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Interestingly, there are now at least 6 cases of gross hematuria reported in patients with a history of biopsy-proven IgA nephropathy (IgAN), involving both mRNA vaccines.1, 2, 3

It has been reported in preclinical trials that **nasal shedding of SARS-CoV-2 still occurred after vaccination with both mRNA vaccines, suggesting a lack of a mucosal IgA response**.1,4

https://www.kidney-international.org/article/S0085-2538(21)00739-0/fulltext

Is The Spike Protein Toxic?

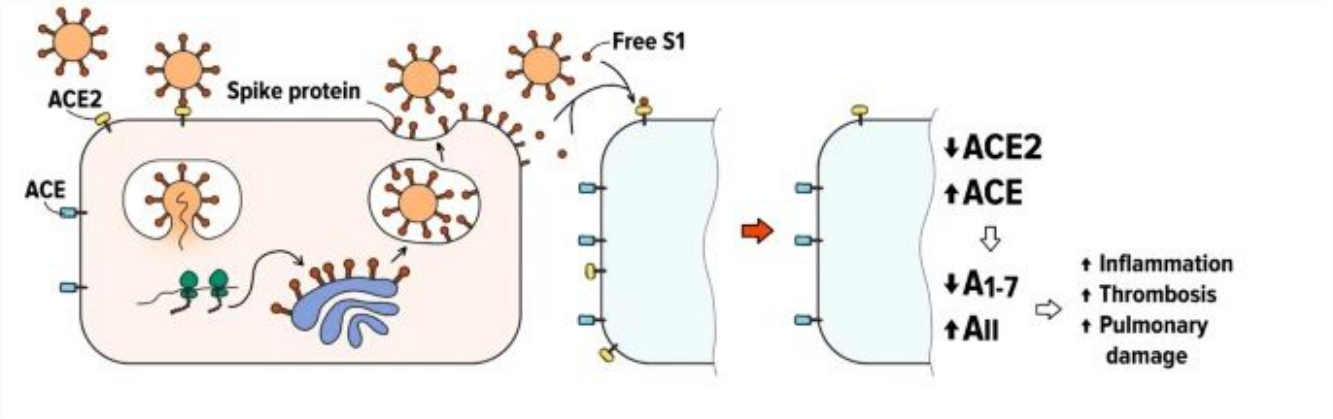
Free SARS-CoV-2 Spike Protein S1 Particles May Play a Role in the Pathogenesis of COVID-19 Infection

We hypothesize that the soluble S1 subunits of the SARS-CoV-2 **S protein shed from the infected cells** and from the virions *in vivo* may bind to the ACE2 and downregulate cell surface expression of this protein. **The decrease in the ACE2 activity on the background of constant or increased ACE activity in the lungs may lead to the prevalence of angiotensin II effects over those of angiotensin (1-7), thus promoting thrombosis, inflammation, and pulmonary damage.**

https://link.springer.com/article/10.1134/S0006297921030032

Is The Spike Protein Toxic?

Figure.



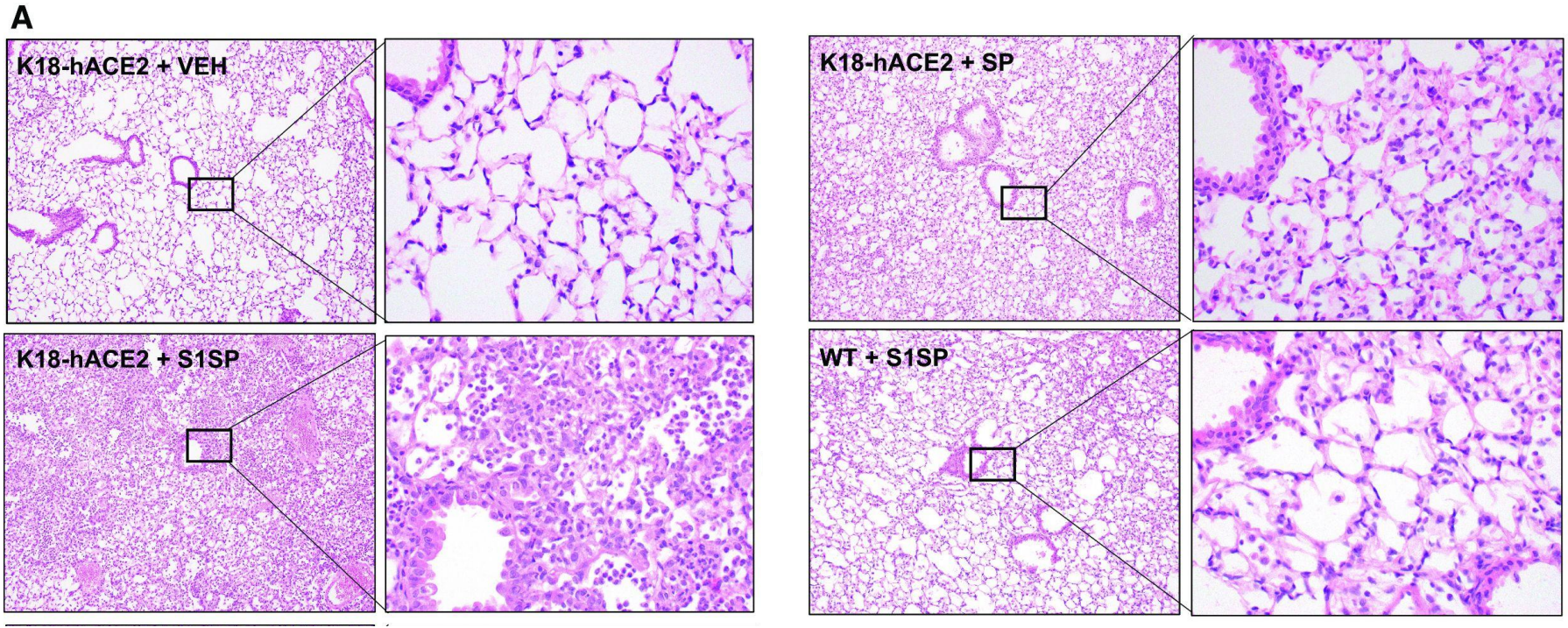
Putative involvement of free S1 subunits of the SARS-CoV-2 S protein in the COVID-19 infection. Spontaneous "firing" of the S protein trimers on the surface of virions and infected cells liberates free RBD-containing S1 particles. The binding of these S1 particles to ACE2 may cause a decrease in the ACE2 cell surface expression and lead to the RAS imbalance.

Is The Spike Protein Toxic?

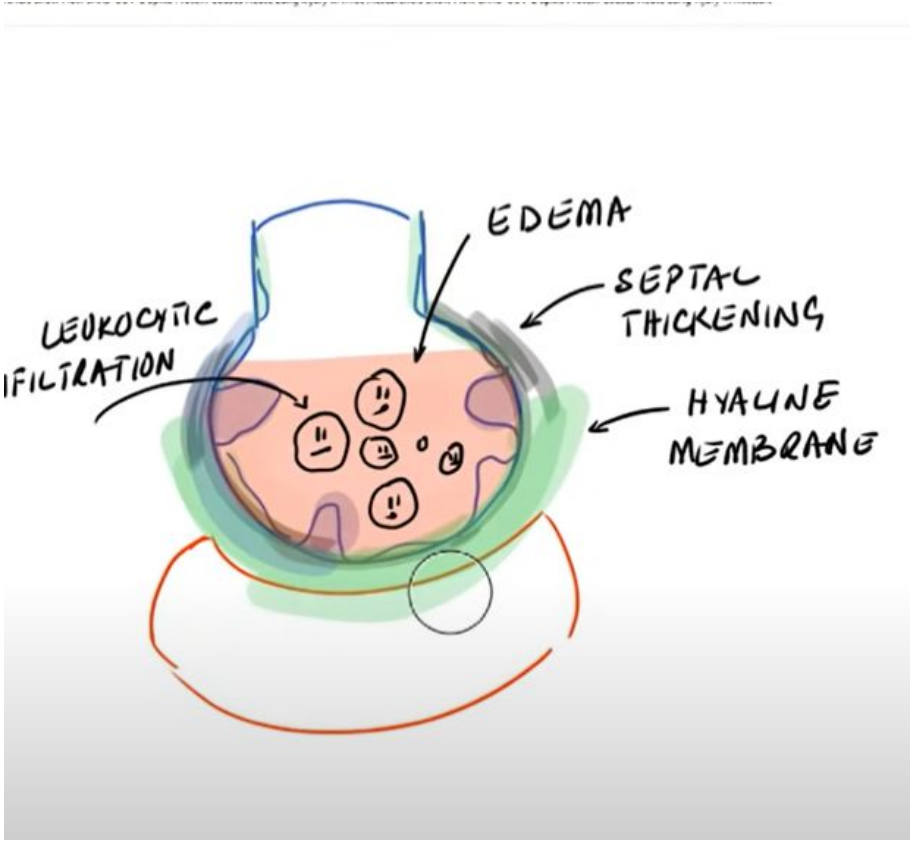
The SARS-CoV-2 spike protein subunit S1 induces COVID-19-like acute lung injury in K18-hACE2 transgenic mice and barrier dysfunction in human endothelial cells

To that end, we intratracheally instilled the S1 subunit of SARS-CoV-2 spike protein (S1SP) in K18-hACE2 transgenic mice that overexpress human ACE2 and examined signs of COVID-19-associated lung injury 72 h later. K18-hACE2 mice instilled with S1SP exhibited a decline in body weight, dramatically increased white blood cells and protein concentrations in bronchoalveolar lavage fluid (BALF), upregulation of multiple inflammatory cytokines in BALF and serum, histological evidence of lung injury, and activation of signal transducer and activator of transcription 3 (STAT3) and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) pathways in the lung. K18-hACE2 mice that received either saline or SP exhibited little or no evidence of lung injury. WT mice that received S1SP exhibited a milder form of COVID-19 symptoms, compared with the K18-hACE2 mice. Furthermore, S1SP, but not SP, decreased cultured human pulmonary microvascular transendothelial resistance (TER) and barrier function.

https://pubmed.ncbi.nlm.nih.gov/34156871/


A
K18-hACE2 + VEH
K18-hACE2 + SP
K18-hACE2 + S1SP
WT + S1SP

Is The Spike Protein Toxic?



Is The Spike Protein Toxic?

SARS-CoV-2 spike protein S1 subunit induces pro-inflammatory responses via toll-like receptor 4 signaling in murine and human macrophages

To elucidate the inflammatory mechanisms involved in COVID-19, we examined the effects of SARS-CoV-2 spike protein S1 subunit (hereafter S1) on the pro-inflammatory responses in murine and human macrophages. Murine peritoneal exudate macrophages produced pro-inflammatory mediators in response to S1 exposure. Exposure to S1 also activated nuclear factor-κB (NF-κB) and c-Jun N-terminal kinase (JNK) signaling pathways. Pro-inflammatory cytokine induction by S1 was suppressed by selective inhibitors of NF-κB and JNK pathways.

These results suggest that SARS-CoV-2 spike protein S1 subunit activates TLR4 signaling to induce pro-inflammatory responses in murine and human macrophages. Therefore, TLR4 signaling in macrophages may be a potential target for regulating excessive inflammation in COVID-19 patients.

It is possible that S1 binds to ACE2, which mediates pro-inflammatory responses in macrophages.

https://www.sciencedirect.com/science/article/pii/S2405844021002929

Is The Spike Protein Toxic?

The Effects of Aß1-42 Binding to the SARS-CoV-2 Spike Protein S1 Subunit and Angiotensin-Converting Enzyme 2.

Here, our findings demonstrate that Aß1-42, but not Aß1-40, bound to various viral proteins with a preferentially high affinity for the spike protein S1 subunit (S1) of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and the viral receptor, angiotensin-converting enzyme 2 (ACE2). These bindings were mainly through the C-terminal residues of Aß1-42. Furthermore, Aß1-42 strengthened the binding of the S1 of SARS-CoV-2 to ACE2 and increased the viral entry and production of IL-6 in a SARS-CoV-2 pseudovirus infection model.

In conclusion, these findings suggest that the binding of Aß1-42 to the S1 of SARS-CoV-2 and ACE2 may have a negative impact on the course and severity of SARS-CoV-2 infection.

https://pesquisa.bvsalud.org/global-literature-on-novel-coronavirus-2019-ncov/resource/pt/covidwho-1350316

Is The Spike Protein Toxic?

The SARS-CoV-2 Spike protein disrupts human cardiac pericytes function through CD147-receptor-mediated signalling: a potential non-infective mechanism of COVID-19 microvascular disease

Results show, for the first time, that cardiac PCs are not permissive to SARS-CoV-2 infection in vitro, whilst a recombinant S protein alone elicits functional alterations in PCs. This was documented as: (1) increased migration, (2) reduced ability to support endothelial cell (EC) network formation on Matrigel, (3) secretion of pro-inflammatory molecules typically involved in the cytokine storm and (4) production of pro-apoptotic factors responsible for EC death.

In conclusion, our findings suggest that circulating S protein prompts vascular PC dysfunction, potentially contributing to establishing microvascular injury in organs distant from the site of infection. This mechanism may have clinical and therapeutic implications.

We provide evidence that cardiac PCs are not infected by SARS-CoV-2. Importantly, we show that the recombinant S protein alone elicits cellular signalling through the CD147 receptor in cardiac PCs, thereby inducing cell dysfunction and microvascular disruption in vitro. **This study suggests that soluble S protein can potentially propagate damage to organs distant from sites of infection, promoting microvascular injury.** Blocking the CD147 receptor in patients may help protect the vasculature not only from infection, but also from the collateral damage caused by the S protein.

REF: https://www.biorxiv.org/content/10.1101/2020.12.21.423721v2.full.pdf

Is The Spike Protein Toxic?

The S1 protein of SARS-CoV-2 crosses the blood–brain barrier in mice

Mechanistic studies indicated that **I-S1 crosses the blood–brain barrier** by adsorptive transcytosis and that murine **angiotensin-converting enzyme 2** is involved in brain and lung uptake…

All tissues showed uptake of I-S1 (Fig. 2b–f). Spleen and liver uptake was nonlinear, suggesting that their tissue beds were coming into equilibrium with blood. Most substances in blood are cleared by kidney or liver; the **much higher I-S1 uptake in liver compared to kidney suggests that I-S1 is cleared from blood predominantly by the liver.** To determine if there were regional differences in I-S1 uptake within the brain, we collected the olfactory bulb and dissected the whole brain into ten regions (Extended Data Fig. 2). **We found that I-S1 entered all brain regions**, with no statistically significant differences among them.

https://www.nature.com/articles/s41593-020-00771-8

Is The Spike Protein Toxic?

Cell death and pathological findings of the spleen in COVID-19 patients

It was found that up to 67% of these immune cells were positive for spike protein.

SARS-CoV-2 uses it's spike protein(S protein) to bind with the angiotensin-converting enzyme 2 (ACE2) receptor on target cells.

We then sought to explore whether SARS-CoV-2 could attack immune cells and macrophages.

The data presented showed that SARS-CoV-2 S protein and CD11b co-expression were readily detectable in these tissues from COVID-19 patients(Fig. 4B). The SARS-CoV-2 S protein-positive rate among CD11b positive cells was up to 67%(Fig. 4E). In addition, cells of spleen tissue from COVID-19 patients were also successively co-stained with SARS-CoV-2 S protein and CD68 antibodies(Fig. 4 C). The SARS-CoV-2 S protein-positive rate among CD68 positive cells was up to 68.1%(Fig. 4 F).

Reports of elevated cytokine levels and beneficial effects of immunosuppressant agents in COVID-19 patients suggest that **the pathogenesis of COVID-19 may be related to cytokine storms** [18], [19]. Studies also showed that cytokine storms were associated with poor outcomes[20]. However, previous studies of COVID-19 have done little on the role of the spleen in the pathogenesis of cytokine storm although the spleen is the largest secondary lymphoid organ in the body and as such hosts a wide range of immunological functions[12]. In this study, we have found that (1) COVID-19 patients have a higher rate of apoptotic and dead cells in spleen tissue than that of non-COVID-19 control patients; (2) **there were more immune cells including macrophages in COVID-19 patients compared to the control group within the spleen tissue, indicating that infiltrating immune cells may play an important role in the pathogenesis of COVID-19;**

REF: https://www.sciencedirect.com/science/article/pii/S0344033821002715

Is The Spike Protein Toxic?

Circulating Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Vaccine Antigen Detected in the Plasma of mRNA-1273 Vaccine Recipients

Authors detected S1 and N in 64% of COVID-19 positive patients, and **S1 levels were significantly associated with disease severity.**

After the first 100-μg dose, the mRNA-1273 vaccine produced detectable levels of **S1 antigen in plasma in 11 participants**, **and spike antigen was detected in 3 of 13 participants**. **Nucleocapsid antigen was undetectable** or at background levels in all participants after both injections, as expected.

S1 antigen was detected as early as day 1 postvaccination, and peak levels were detected on average 5 days after the first injection (Figure 1A). S1 in all participants declined and became undetectable by day 14.

In all 13 participants, as expected, IgG levels against spike, S1, and RBD increased after the first injection, whereas IgG against nucleocapsid showed no change over time. IgA is involved in early neutralization activity and is therefore crucial to target potentially short-lived IgA responses [8]. Our Simoa assays detected increased IgA against spike, S1, and RBD after the first injection.

REF: https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab465/6279075

Is The Spike Protein Toxic?

Virological and Serological Characterization of SARS-CoV-2 Infections Diagnosed After mRNA BNT162b2 Vaccination

"Most cases (78%) showed infection in presence of neutralizing antibodies at the time of infection diagnosis, presumably attributable to vaccination, **due to the concomitant absence of anti-N IgG in most cases." Proof that the vaccine only produces antibodies to the spike and not the nucleocapsid.**

https://www.medrxiv.org/content/10.1101/2021.09.21.21263882v1.full

What are the effects of the vaccines on innate immunity?

Mucosal Immunity in Covid-19: A Neglected, But Critical Aspect of SARS-CoV-2 Infection

“There is a significant role for mucosal immunity and for secretory as well as circulating IgA antibodies in COVID-19.”

As SARS-CoV-2 first mainly infects the upper respiratory tract (URT), mucosal immune responses are expected to be induced in the nasopharynx, both across the nasal epithelium and *via* the tonsils and adenoids, which are collectively referred to as nasopharynx-associated lymphoid tissue (NALT) that serve as inductive sites for the mucosal immune system (6, 7).

The serious pathology of COVID-19 occurs in the terminal airways of the lungs, where circulating IgG is the dominant immunoglobulin. The resulting **intense inflammation** involves multiple molecular and cellular factors, including cells recruited by virus-induced chemo-attractants (17).

SIgA is essentially non-inflammatory, even anti-inflammatory, in its mode of action.

Selective **IgA deficiency** affects both mucosal and circulatory compartments and **subjects often show increased susceptibility to URT infections**. If mucosal SIgA antibodies in the URT exert a protective effect against the early stages of SARS-CoV-2 infection, then **deficiency of SIgA would be expected to enhance the infection, facilitating descent into the LRT and leading to advanced disease.**

https://www.frontiersin.org/articles/10.3389/fimmu.2020.611337/full

What are the effects of the vaccines on innate immunity?

Human IgG and IgA responses to COVID-19 mRNA vaccines

In summary, longitudinal serology of COVID-19 mRNA vaccine recipients highlights important issues related to immunity and monitoring of vaccine responses. The persistence of spike antigen-specific serum IgG following vaccination is hopefully a positive indicator of effective long-lived immunity, and clinical indicator of vaccine responsiveness [27]. In addition to IgG, the data demonstrate COVID-19 mRNA vaccines also elicit antigen-specific IgA, which may be important in preventing transmission as well as infection [28,29]. **Spike-specific serum IgA levels decay significantly ($p < 0.002$) faster than spike-specific IgG**, however, the “recall” response for both IgG and IgA (time to peak serum levels following the 2nd / booster dose) is significantly ($p < 0.03$) shorter than the primary response.

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0249499

What are the effects of the vaccines on innate immunity?

First known case of postmortem study in a patient vaccinated against SARS-CoV-2

Dr. Robert Gorter:

A major concern is that these experimental **vaccinations containing mRNA (spike proteins) could well inhibit or even wipe out innate as well as the adaptive immunity.**

Therefore, my group and I demand a halt to applying these experimental vaccines world-wide and, for sure, abort each initiative to vaccinate children and infants.

http://robert-gorter.info/first-known-case-postmortem-study-patient-vaccinated-sars-cov-2/

What are the effects of the vaccines on innate immunity?

Acute kidney injury with gross hematuria and IgA nephropathy after COVID-19 vaccination

It has been reported in preclinical trials that nasal shedding of SARS-CoV-2 still occurred after vaccination with both mRNA vaccines, suggesting a lack of a mucosal IgA response.1,4

https://www.kidney-international.org/article/S0085-2538(21)00739-0/fulltext

What are the effects of the vaccines on innate immunity?

COVID-19 Vaccines May Not Prevent Nasal SARS-CoV-2 Infection and Asymptomatic Transmission

Systemic respiratory vaccines generally provide limited protection against viral replication and shedding within the airway, as this requires a local mucosal secretory IgA response. Indeed, preclinical studies of adenovirus and mRNA candidate vaccines demonstrated persistent virus in nasal swabs despite preventing COVID-19.

https://journals.sagepub.com/doi/full/10.1177/0194599820982633

What are the effects of the vaccines on innate immunity?

Rogue Antibodies Involved In Nearly One-Fifth of Covid Deaths

Around 10% of people with severe COVID-19 had **autoantibodies that attack and block type 1 interferons, protein molecules in the blood that have a critical role in fighting off viral infections.**

The international research team focused on detecting autoantibodies that could neutralize lower, more physiologically relevant concentrations of interferons. They studied 3,595 patients from 38 countries with critical COVID-19, meaning that the individuals were ill enough to be admitted to an intensive-care unit. **Overall, 13.6% of these patients possessed autoantibodies, with the proportion ranging from 9.6% of those below the age of 40, up to 21% of those over 80. Autoantibodies were also present in 18% of people who had died of the disease.**

https://www.scientificamerican.com/article/rogue-antibodies-involved-in-nearly-one-fifth-of-covid-deaths1/

What Do The Autopsies Show?

First Case of Post Mortem Study In A Patient Vaccinated With SARS-CoV-2

Spike protein **(S1)** antigen-binding showed **significant levels** for immunoglobulin (Ig) G, while **nucleocapsid IgG/IgM was not elicited**. Postmortem molecular mapping by real-time polymerase chain reaction revealed relevant SARS-CoV-2 cycle threshold values in all organs examined (oropharynx, olfactory mucosa, trachea, lungs, heart, kidney and cerebrum) except for the liver and olfactory bulb. These results might suggest that the **first vaccination induces immunogenicity but not sterile immunity.**

We demonstrated viral RNA in nearly all organs examined except for the liver and the olfactory bulb (Figure 1).

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8051011/

What Do The Autopsies Show?

COVID-19 pulmonary pathology: a multi-institutional autopsy cohort from Italy and New York City

“Immunohistochemistry was carried out on selected cases to characterize the platelet component of microthrombi (CD61 clone 2F2, Leica Biosystems, IL) and to identify **SARS-CoV-2 viral spike protein** (Genetex clone 1A9 at 1:75 dilution with 20-min antigen retrieval at pH 9.0 on Leica Bond III automated instrument). **SARS-CoV-2 RNA was detected in trachea and lung** by RNAscope® technology (Advanced Cell Diagnostics, Newark, CA), using SARS-CoV-2 2019-S (cat. 848561), for detection of **viral spike protein-encoding RNA**. RNA integrity was assessed using a probe targeting the *UBC* (Ubiquitin C) housekeeping gene. Overall, 23 cases were examined for spike protein IHC, and all positives were confirmed using RNA in situ for **spike protein-encoding RNA**.”

https://www.nature.com/articles/s41379-020-00661-1

What Do The Autopsies Show?

Autopsy Findings In 32 Patients With Covid-19: A Single Institution Experience

“Results: SARS-CoV-2 infection was confirmed by nasopharyngeal RT-PCR in 31 cases (97%) and by immunohistochemical staining for **SARS-CoV-2 spike protein in the lung** in the remaining 1 case (3%).” **Only spike protein was found in the lungs, not the virus.**

https://www.karger.com/Article/Pdf/511325

What Do The Autopsies Show?

Multiorgan tropism of SARS-CoV-2 lineage B.1.1.7

We speculate that B.1.1.7 spike protein's affinity to human ACE2 facilitates transmission, organ tropism, and ultimately morbidity and mortality. Our results indicate that also SARS-CoV-2 B.1.1.7 has a relevant organ tropism beyond the respiratory tract. **We speculate that B.1.1.7 spike protein's affinity to human ACE2 facilitates transmission, organ tropism, and ultimately morbidity and mortality**.

https://link.springer.com/article/10.1007/s00414-021-02691-z

What Do The Autopsies Show?

A cohort autopsy study defines COVID-19 systemic pathogenesis

Through systemic autopsy examination, we found that **SARS-CoV-2 RNA, spike protein** or virion-like particles existed in the lungs and multiple extrapulmonary organs in critically ill patients as long as 15–67 days after symptom onset. The SARS-CoV-2 viral **RNA distributed in postmortem organs including those in the respiratory, digestive, genitourinary, cardiovascular, immune systems, endo/exocrine glands, and skin (Fig. 1b, c)**.

Pulmonary areas with higher expression of **SARS-CoV-2 spike protein** were featured by **hyperproliferation of epithelia** (Supplementary information, **Fig. S2b, c)**. Further analyses revealed that the proliferative cells containing SARS-CoV-2 were mainly ACE2-expressing and TTF-1-positive **alveolar epithelia and bronchiolar basal cells** (Supplementary information, **Fig. S2d, e)**.

Remarkably, in ten COVID-19 patients with medical records of respiratory failure, mechanical ventilation, and arterial oxygen partial pressure (PaO2), **mucus plugs were present in the alveoli or bronchioles (Fig. 2g)**, which were inversely associated with the levels of PaO2 **(Fig. 2h)**, suggesting that **mucus production was increased in hypoxemia and may limit the efficacy of mechanical ventilation during COVID-19 treatment**.

We found that **SARS-CoV-2 spike protein** was present in **CD34+ endothelia at blood–air barrier or pulmonary vessels in serial sections of the COVID-19 lungs (Fig. 3a), raising the possibility that SARS-CoV-2 was able to infiltrate blood–air barrier for intrapulmonary and systemic dissemination. SARS-CoV-2 spike protein was mainly detected in the glomeruli with abundant endothelium-formed filtration barriers and renal proximal convoluted tubular epithelia in the kidneys**

SARS-CoV-2 spike protein was also detected in endothelia of the **blood–testis barrier (Fig. 3c)**, spermatogenic cells and stromal cells in the seminiferous tubules, and sperms in the epididymis in the COVID-19 testes positive for SARS-CoV-2 RNA (Cases 2, 5 and 11) **(Fig. 3c)**. These results provide evidence of SARS-CoV-2 presence in the endothelia including those in physiological barriers (blood–air, blood–testis, and filtration barriers), implying that these cells are susceptible to SARS-CoV-2 infection followed by systemic dissemination.

We found that the cellular **components of alveolar exudate were mainly CD68+ macrophages positive for SARS-CoV-2 spike protein (Fig. 4a)**. IHC staining using serial sections also identified the presence of SARS-CoV-2 spike protein in monocytes and macrophages in lymph nodes and the spleen **(Fig. 4b, c)**, as well as peripheral blood mononuclear cells in the postmortem lungs, kidneys, lymph nodes, spleen, and intestines **(Fig. 4d)**.

What Do The Autopsies Show?

SARS-CoV-2 infection of the central nervous system in a 14-month-old child: A case report of a complete autopsy

The SARS-CoV-2 spike protein has been demonstrated in cortical neurons and in cerebrovascular endothelium [9]. Although detection of SARS-CoV-2 RNA in cerebrospinal fluid (CSF) is uncommon, it has been reported in two adults [10] and one infant [11].

The brain exhibited severe atrophy and neuronal loss. SARS-CoV-2 spike protein (SP) was demonstrated by immunostaining along the ChP epithelium and ependymal cells of the lateral ventricle, and in ChP capillaries and vessels.

https://www.sciencedirect.com/science/article/pii/S2667193X21000387

Can mRNA Be Converted Into DNA?

Reverse-transcribed SARS-CoV-2 RNA can integrate into the genome of cultured human cells and can be expressed in patient-derived tissues

We used three different approaches to detect genomic SARS-CoV-2 sequences integrated into the genome of infected cells. These approaches were Nanopore long-read sequencing, Illumina paired-end whole genomic sequencing, and Tn5 tagmentation-based DNA integration site enrichment sequencing. **All three methods provided evidence that SARS-CoV-2 sequences can be integrated into the genome of the host cell.**
These results suggest that SARS-CoV-2 RNA can be reverse-transcribed, and the resulting DNA could be integrated into the genome of the host cell.

Table 1 summarizes all of the linked SARS-CoV-2–host sequences that were recovered. **DNA copies of portions of the viral genome were found in almost all human chromosomes.**

However, if a cell with an integrated and expressed SARS-CoV-2 sequences survives and presents a viral- or neo-antigen after the infection is cleared, **this might engender continuous stimulation of immunity without producing infectious virus and could trigger a protective response or conditions such as autoimmunity** as has been observed in some patients (**62**, **63**). The presence of LCMV sequences integrated in the genomes of acutely infected cells in mice led the authors to speculate that expression of such sequences “potentially represents a **naturally produced form of DNA vaccine**” (**30**).

Derepressed LINE1 expression, induced by viral infection or by exposure to cytokines (**38⇓–40**), **may stimulate SARS-CoV-2 integration into the genome of infected cells in patients.**

https://www.pnas.org/content/118/21/e2105968118

Can mRNA Be Converted Into DNA?

Exogenous Coronavirus Interacts With Endogenous Retrotransposon in Human Cells

Increased retrotransposon RNA may further form chimeric transcripts with coronavirus RNA, and **integrate viral genomic fragments into human genome.**

Taken together, we demonstrate that **coronavirus infection increases retrotransposon expression in human cells**, possibly through global DNA hypomethylation, and increased retrotransposon RNA may further form chimeric transcripts with coronavirus RNA for **integration of viral genomic fragments into human genome. These enhanced retrotransposon transcripts may be long-term inherited to harm host organs**.

https://www.frontiersin.org/articles/10.3389/fcimb.2021.609160/pdf

Can mRNA Be Converted Into DNA?

Recombination of Retrotransposon and Exogenous RNA Virus Results in Nonretroviral cDNA Integration

We found that illegitimate recombination between an exogenous nonretroviral RNA virus, lymphocytic choriomeningitis virus, and the endogenous intracisternal A-type particle (IAP) retrotransposon occurred and **led to reverse transcription of exogenous viral RNA. The resulting complementary DNA was integrated into the host's genome** with an IAP element. Thus, RNA viruses should be closely scrutinized for any capacity to interact with endogenous retroviral elements before their approval for therapeutic use in humans.

https://www.science.org/doi/10.1126/science.1167375