Is The Vaccine Safe and Effective?

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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 early 2023:**

Currently given emergency approval (EUA), Conditional Marketing Authorisation (CMA) etc all these terms mean they are only being used because of an "emergency" situation. These injections have not been fully licensed anywhere in the world, with the exception of the Comirnaty vaccine, however, that vaccine has proven ineffective at preventing SARS-CoV-2, thus the EUA of the Pfizer booster shot.

Pfizer Trials :Pfizer Trials https://clinicaltrials.gov/ct2/show/NCT04368728
Pfizer example short term approval terms : Pfizer Approval Terms
https://www.fda.gov/media/144414/download

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

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
Cats: https://www.jstage.jst.go.jp/article/jvms/60/1/60_1_49/_article
Mice : https://pubmed.ncbi.nlm.nih.gov/22536382/
Mice: https://pubmed.ncbi.nlm.nih.gov/17194199/
Mice: https://pubmed.ncbi.nlm.nih.gov/18941225/

The trials were short and flawed:

More information on the vaccines is shown in the links below:
Pfizer Trials
https://uploads-ssl.webflow.com/5fa5866942937a4d73918723/6018018e4b1729f3251e4281_UKMFA_Pfizer_COVID-19_Vaccine_(Public1-2).pdf

Covid-19 Injection information leaflet
https://uploads-ssl.webflow.com/5fa5866942937a4d73918723/6006c4e4ccf7a9074538c6ad_UKMFA_COVID-19_Vaccine_Patient_Information.pdf

What is the latest scientific evidence saying on the efficacy/ effectiveness of these Vaccines:

Absolute Risk Reduction i.e. the true impact that the injection itself was shown to have at reducing your chances of getting sick with Covid-19 was just 0.84% for Pfizer and 1.28% for AZ (per the Lancet study linked below).

Link https://www.thelancet.com/journals/lanmic/article/PIIS2666-5247(21)00069-0/fulltext

The injections have numerous serious and fatal short term side effects and no long term safety data

Link Adverse Reaction
:https://www.gov.uk/government/publications/coronavirus-covid-19-vaccine-adverse-reactions

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."

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.
- **Previous clinical (based on COVID-19 symptoms/signs alone, if a SARS-CoV-2 NAAT result was not available) or microbiological (based on COVID-19 symptoms/signs and a positive SARS-CoV-2 NAAT result) 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.**
- Participation in other studies involving study intervention within 28 days prior to study entry through and including 6 months after the last dose of study intervention, with the exception of non-Pfizer interventional studies for prevention of COVID 19, which are prohibited throughout study participation.
- **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.**
- Phase 1 only: Any screening hematology and/or blood chemistry laboratory value that meets the definition of a ≥ Grade 1 abnormality.
- Phase 1 only: Positive test for HIV, hepatitis B surface antigen (HBsAg), hepatitis B core antibodies (HBc Abs), or hepatitis C virus antibodies (HCV Abs) at the screening visit.
- Phase 1 only: SARS-CoV-2 NAAT-positive nasal swab within 24 hours before receipt of study intervention.

- Investigator site staff or Pfizer employees directly involved in the conduct of the study, site staff otherwise supervised by the investigator, and their respective family members.

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

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/

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/

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/**

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

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

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/

Is The Spike Protein Toxic?

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

Intravenous Injection of Coronavirus Disease 2019 (COVID-19) mRNA Vaccine Can Induce Acute Myopericarditis in Mouse Model

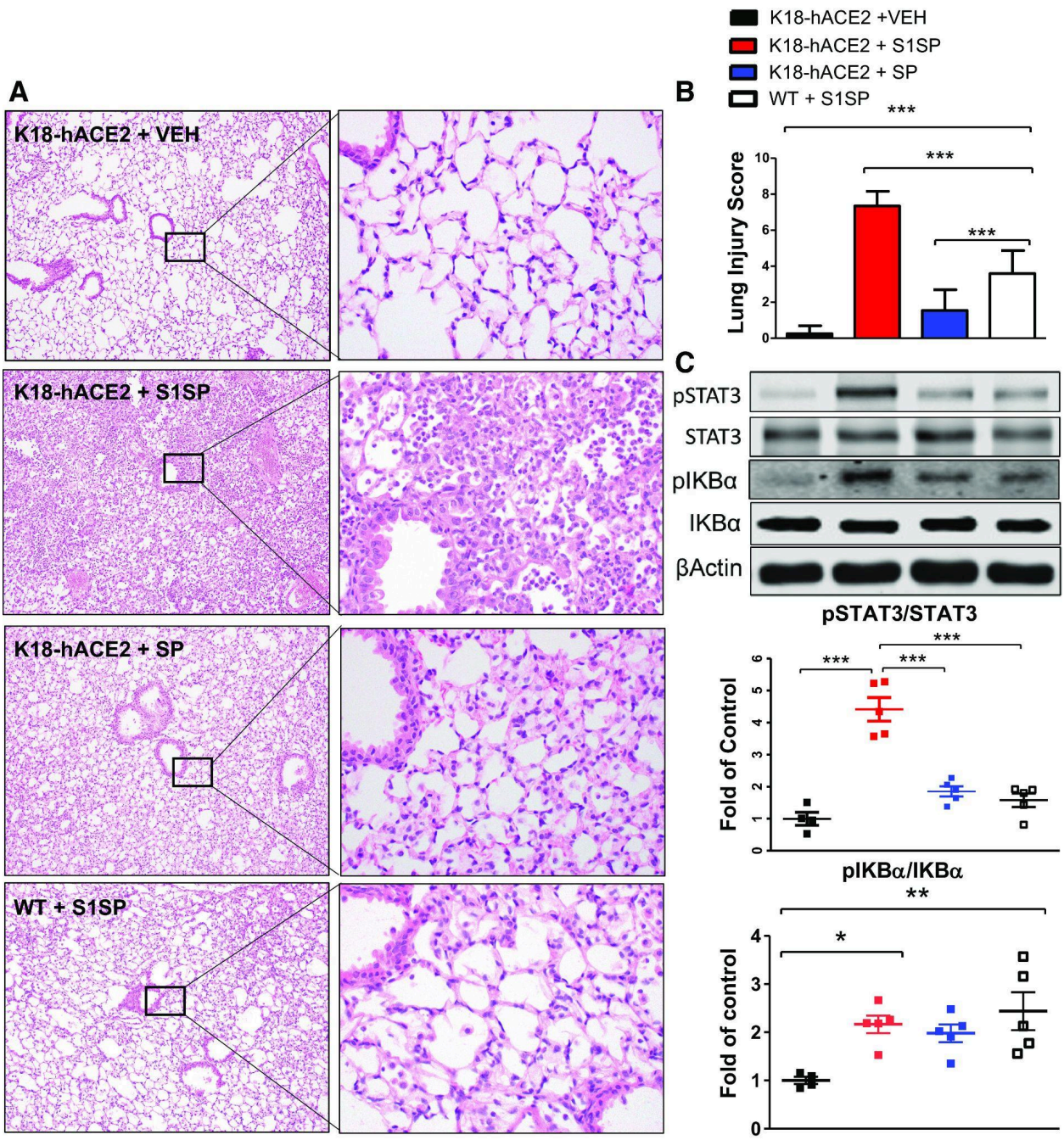
https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab707/6353927

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 + S1SP
K18-hACE2 + SP
WT + S1SP
B
K18-hACE2 +VEH
K18-hACE2 + S1SP
K18-hACE2 + SP
WT + S1SP
***
***
***
Lung Injury Score
0
2
4
6
8
10
C
pSTAT3
STAT3
pIKBα
IKBα
βActin
pSTAT3/STAT3
***
***
***
Fold of Control
0
1
2
3
4
5
6
pIKBα/IKBα
**
*
Fold of control
0
1
2
3
4

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.

SARS-CoV-2 spike protein S1 subunit induces production of pro-inflammatory mediators in murine macrophages

SARS-CoV-2 spike protein S1 subunit activates NF-κB and stress-activated MAPK signaling pathways in murine macrophages

SARS-CoV-2 spike protein S1 subunit induces production of pro-inflammatory cytokines via NF-κB and JNK pathways in murine macrophages

SARS-CoV-2 spike protein S1 subunit induces pro-inflammatory responses via TLR4 signaling in murine and human macrophages

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

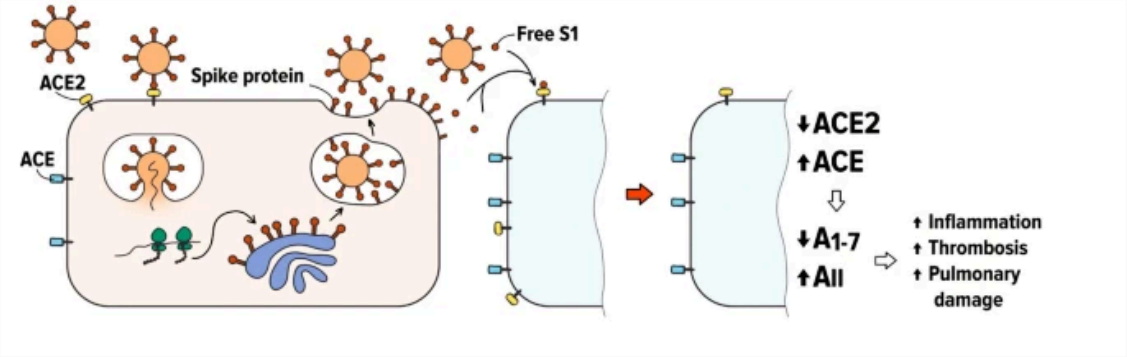
https://www.sciencedirect.com/science/article/pii/S2405844021002929

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

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.

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

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

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

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

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

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?

Why Is The Decline In IgA Levels Alarming?

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

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

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

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

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/

Sterile Immunity the type of immunity that completely prevents a disease-causing pathogen like COVID-19 from establishing an infection.

Unless a vaccine offers sterilizing immunity, there is a chance that the virus can be passed to others even if the infected person has no symptoms.

https://www.verywellhealth.com/covid-19-vaccines-and-sterilizing-immunity-5092148

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

Dr. Robert Gorter:

So far, what is the take-home of this study in connection with several other *post-mortem* studies where viral RNA is found in all organs throughout the body four to 12 weeks after the vaccination against SARS-CoV-2? In all traditional vaccine studies, this has never been documented and **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.**

The Innate vs. Adaptive Immune Response

The first line of defense against non-self pathogens is the innate, or non-specific, immune response. The innate immune response consists of physical, chemical, and cellular defenses against pathogens. The main purpose of the innate immune response is to immediately prevent the spread and movement of foreign pathogens throughout the body.

The second line of defense against non-self pathogens is called the adaptive immune response. Adaptive immunity is also referred to as acquired immunity or specific immunity and is only found in vertebrates. The adaptive immune response is specific to the pathogen presented. The adaptive immune response is meant to attack non-self pathogens but can sometimes make errors and attack itself. When this happens, autoimmune diseases can develop (e.g., lupus, rheumatoid arthritis).

The hallmark of the adaptive immune system is the clonal expansion of lymphocytes. Clonal expansion is the rapid increase of T and B lymphocytes from one or a few cells to millions. Each

clone that originates from the original T or B lymphocyte has the same antigen receptor as the original and fights the same pathogen.

While the innate immune response is immediate, the adaptive immune response is not. However, the effect of the adaptive immune response is long-lasting, highly specific, and is sustained long-term to life-long by memory T cells.

All the documented immediate and long-term side effects are massive and often fatal. **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.**

And, as a last remark, SARS-CoV-2 is ***not*** that fatal viral infection with any known therapies to reward a pandemic status and an emergency approval for experimental vaccines which have never been studied at all in humans, for one year by the FDA and EMA.

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

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

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

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

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)**.

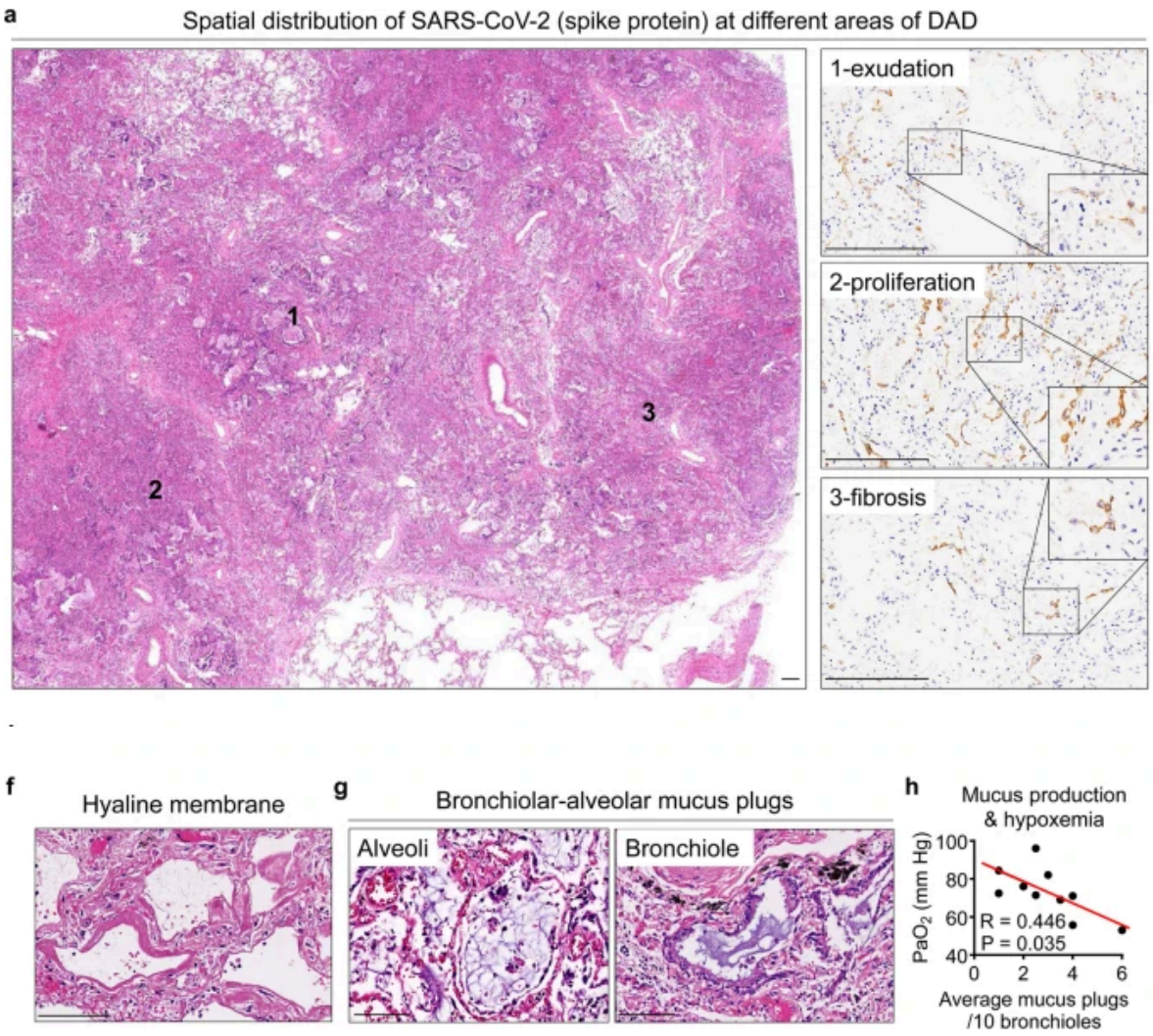
Remarkably, in ten COVID-19 patients with medical records of respiratory failure, mechanical ventilation, and arterial oxygen partial pressure (PaO2), **mucus plugs were present in the alveoli or bronchioles (Fig. 2g)**, which were inversely associated with the levels of PaO2 **(Fig. 2h)**, suggesting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

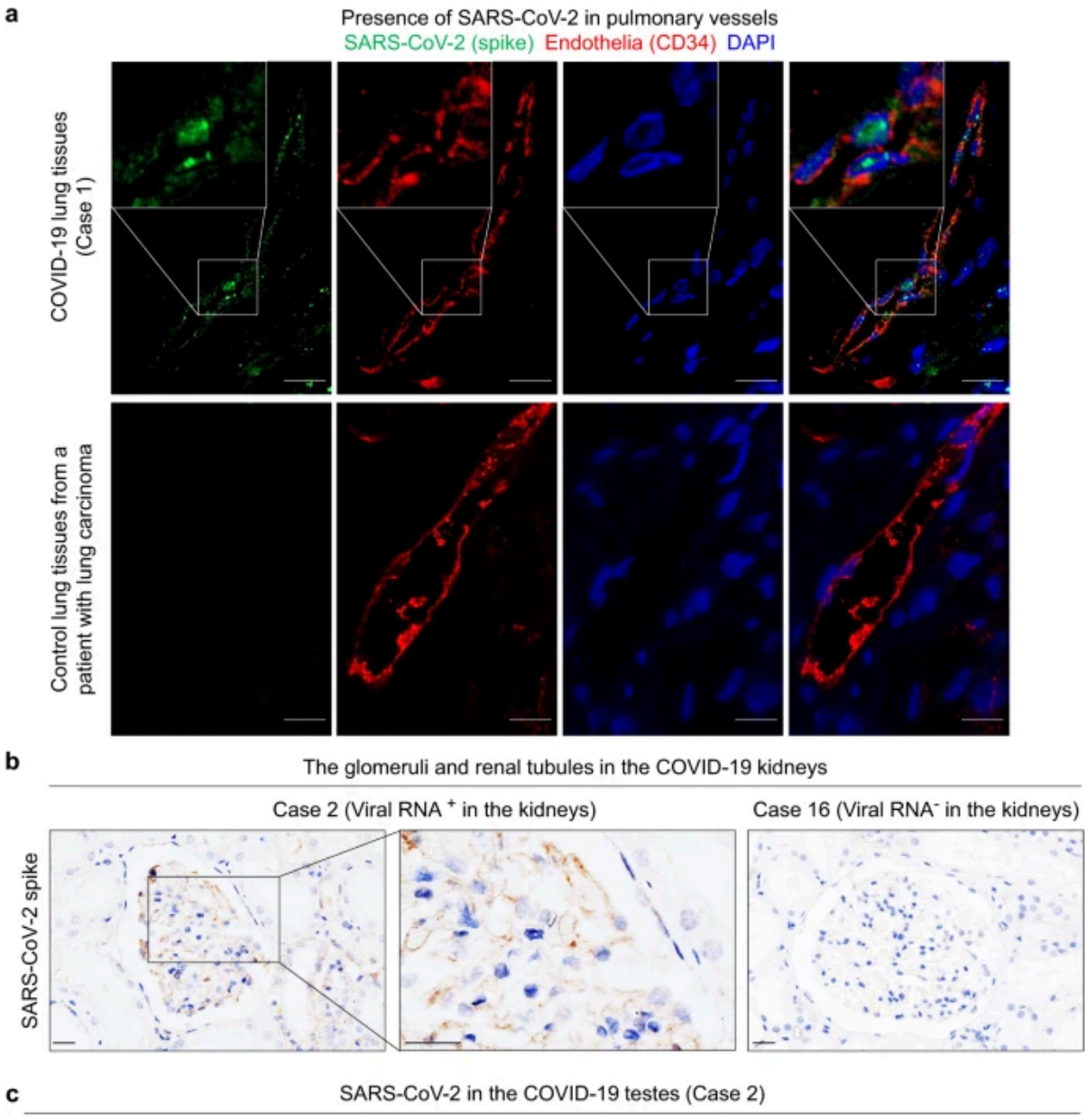
We found that the cellular components of alveolar exudate were mainly CD68+ macrophages positive for SARS-CoV-2 spike protein **(Fig. 4a)**. IHC staining using serial sections also identified the presence of SARS-CoV-2 spike protein in monocytes and macrophages in lymph nodes and the spleen **(Fig. 4b,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

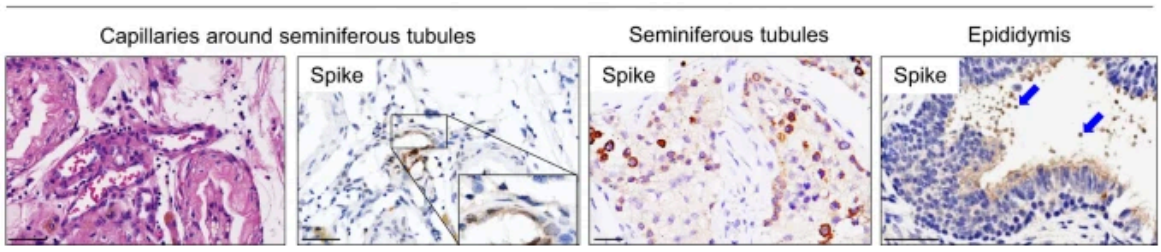
Fig. 2: SARS-CoV-2-associated pulmonary pathological changes.



a H&E and IHC staining showing SARS-CoV-2 spike protein in pulmonary areas manifesting different features (1, exudation; 2, proliferation; 3, fibrosis) of diffuse alveolar damage (DAD). Scale bars, 250 μm. **b**–**e** Proportion of DAD-exudation areas (**b**), DAD-proliferation areas (**c**), and DAD-fibrosis areas (**d**), and the average SARS-CoV-2 RNA (**e**) in postmortem lungs from 15 COVID-19 autopsy cases. **f, g** H&E staining showing hyaline membrane formation (**f**) and bronchiolar-alveolar mucus (**g**). Scale bars, 100 μm. **h** The correlation between average bronchiolar-alveolar mucus plug number and PaO_2 level in patients with respiratory failure.

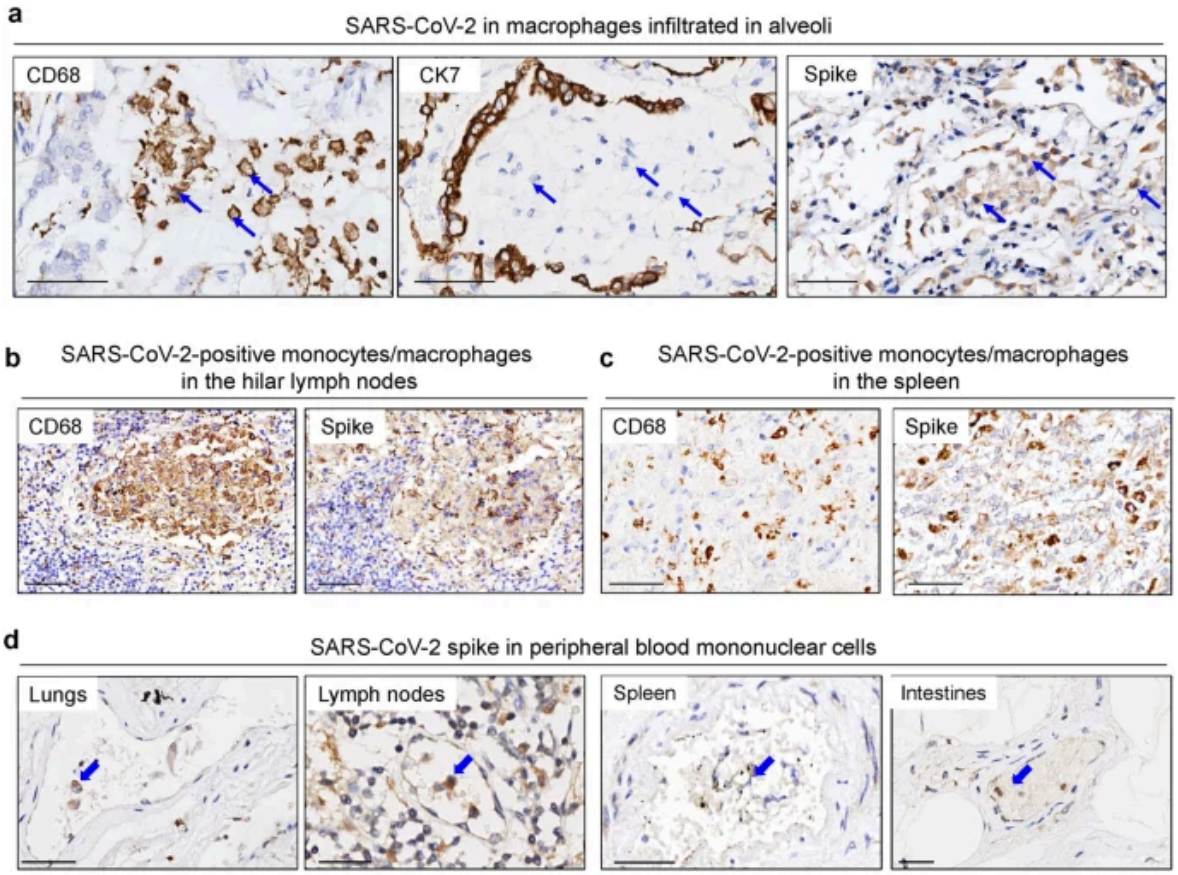
Fig. 3: The presence of SARS-CoV-2 in the endothelia of physiological barriers in the lungs, kidneys, and testes.





a Immunofluorescent staining of SARS-CoV-2 spike protein and CD34 in endothelia of pulmonary vessels using COVID-19 lung tissues (upper panel, Case 1) or control lung tissues from a patient with lung carcinoma (lower panel). Scale bars, 25 μm. **b** IHC showing that SARS-CoV-2 spike protein was detected in glomeruli with abundant filtrated barriers and convoluted tubular epithelia in the kidneys positive for viral RNA (Case 2). The kidney tissues (Case 16) negative for viral RNA were used as control. Scale bars, 25 μm. **c** H&E staining and IHC staining showing SARS-CoV-2 spike in endothelia of the blood–testis barrier, seminiferous tubules, and sperms in the epididymis (blue arrows) of the testes from COVID-19 patients (Case 2). Scale bars, 50 μm.

Fig. 4: Evidence of the presence of SARS-CoV-2 in circulating and infiltrating monocytes and macrophages.



a IHC staining of CD68, CK7, and viral spike in alveoli on serial sections. Macrophages are indicated by blue arrows. Scale bars, 50 µm. **b**, **c** IHC staining of monocytes/macrophages marked by CD68 and viral spike protein in lymph nodes (**b**) and the spleen (**c**) on serial sections from COVID-19 patients. Scale bars, 50 µm. **d** IHC staining showing viral spike in peripheral blood mononuclear cells (blue arrows) in vessels of the indicated postmortem organs from COVID-19 patients. Scale bar, 50 µm. **e** U-MAP showing scRNA-seq of 1437 cells on COVID-19 autopsy lung tissues (Case 17). $CD8^+$ T, $CD8^+$ T cells; $CD14^+$ Mono-1/2, $CD14^+$ monocyte-1/2; MoAM-1/2, monocyte-derived alveolar macrophages-1/2; AT, alveolar epithelial type 1/2 cells; Erythroid-like, erythroid-like and erythroid precursor cells; EC, endothelial cells; Fibro, fibroblast cells; $MKI67^+$, $MKI67^+$cells; Plasma, plasma cells. **f** Detection of SARS-CoV-2 transcripts. Plot shows SARS-CoV-2 ORF_10 or nucleocapsid (N) genes in $CD14^+$ monocyte-1 from scRNA-seq. **g** U-MAP showing the expression of *BSG* (encoding CD147), *TFRC* (encoding transferrin receptor-1), *NRP1* (encoding neuropilin-1), and *ACE2* in the scRNA-seq of COVID-19 lung tissues.

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

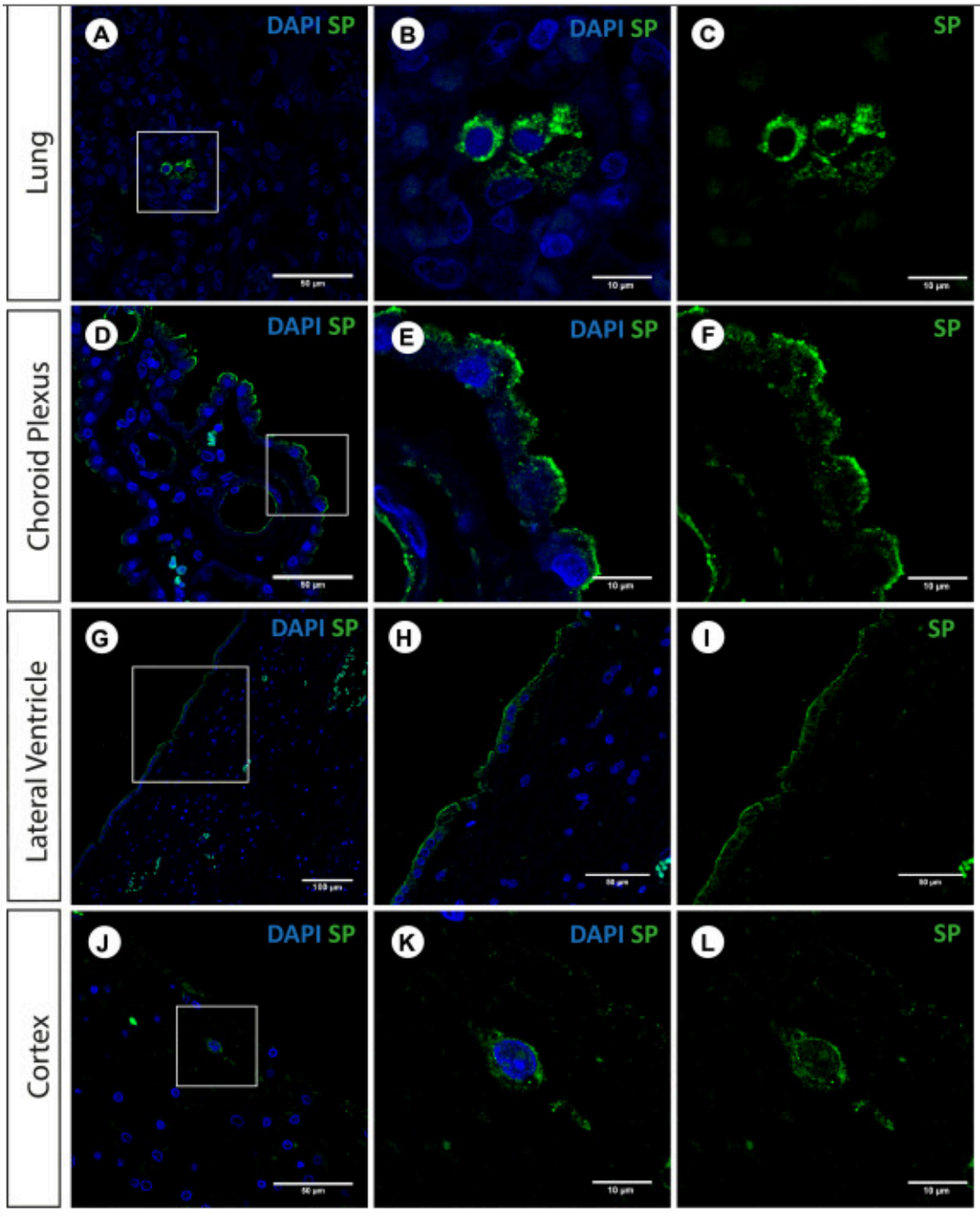


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Figure 2. SARS-CoV-2 detection in lungs, choroid plexus, lateral ventricle and cerebral cortex. Photomicrographs of immunostaining for spike protein in lung tissue (**A-C**) and in the brain (choroid plexus - **D-F**; lateral ventricle - **G-I**; cerebral cortex - **J-L**). Scale bars: (A, D, H, I, J) 50 μm; (G) 100 μm; (B, C, E, F, K, L) 10 μm.

Myocarditis-induced Sudden Death after BNT162b2 mRNA COVID-19 Vaccination in Korea: Case Report Focusing on Histopathological Findings

We present autopsy findings of a 22-year-old man who developed chest pain 5 days after the first dose of the BNT162b2 mRNA vaccine and died 7 hours later. Histological examination of the heart revealed isolated atrial myocarditis, with neutrophil and histiocyte predominance. Immunohistochemical C4d staining revealed scattered single-cell necrosis of myocytes which was not accompanied by inflammatory infiltrates. Extensive contraction band necrosis was observed in the atria and ventricles. There was no evidence of microthrombosis or infection in the heart and other organs. The primary cause of death was determined to be myocarditis, causally-associated with the BNT162b2 vaccine.

https://jkms.org/DOIx.php?id=10.3346/jkms.2021.36.e286

Neuromyelitis Optica in a Healthy Female After Severe Acute Respiratory Syndrome Coronavirus 2 mRNA-1273 Vaccine

Neuromyelitis optica spectrum disorder is an autoimmune demyelinating disease with high relative prevalence in the East Asian population. Clinical manifestations include optic neuritis, longitudinally extensive transverse myelitis, area postrema syndrome, brainstem syndromes, and diencephalic syndromes.

In this case report, we present a case of neuromyelitis optica spectrum disorder that developed 10 days after the first dose of the severe acute respiratory syndrome coronavirus 2 mRNA-1273 vaccine. The patient was a previously healthy White female, completely independent and functional at baseline. She presented with bilateral lower-extremity numbness/tingling, weakness, and urinary retention. Although her neuromyelitis optica IgG was negative, the MRI was consistent with neuromyelitis optica involving and spanning longitudinally the C6-T2 vertebrae. She was treated with IV steroids and her symptoms improved.

Given the novelty of the COVID-19 vaccines and the paucity of literature regarding their adverse effects, case reports such as ours provide unique information that aids healthcare providers in accurately diagnosing and treating patients, ultimately minimizing long-term neurologic deficits.

https://www.ncbi.nlm.nih.gov/labs/pmc/articles/PMC8516014/

A Late Presentation of COVID-19 Vaccine-Induced Myocarditis

With the introduction of the coronavirus disease 2019 (COVID-19) mRNA vaccines, the incidence of severe infection has significantly decreased. While the vaccines have been shown to be effective and safe, there have been few case reports of acute myocarditis within 3-5 days following the second dose of the vaccine. We report a case of an elderly man who presented with acute-onset chest pain after three months of receiving the second dose of the mRNA vaccine. He was found to have acute myocarditis on cardiac magnetic resonance imaging (CMRI), which was attributed to exposure to the COVID-19 vaccine in the absence of any other risk factors. Our patient demonstrated quick resolution of symptoms and was discharged within 72 hours. We review the literature and summarize published case reports on COVID-19 vaccine-associated myocarditis. The present case report provides new evidence regarding the possible subacute presentation of myocarditis post-COVID-19 vaccine, and further highlights the favorable outcome in this newly described clinical entity.

https://www.ncbi.nlm.nih.gov/labs/pmc/articles/PMC8504680/