COV-2 & The Frontal Lobe

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TDBILASH: opinion: COV-2 and the Frontal Lobe:

The brain is the most recent and least studied areas for me, with a massive amount of material. Actual infection, processes prior to COV-2 replication and/or where that replicttion takes place, and pathology from other processes need to be distinguised. Virus can be cultured from just about every body tissue after infection, yet we were assured it stays in the Arm Muscle. That the spike replicates and “infects” (Prions) is a brand new branch of Medicine.

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*-**“SARS-CoV-2 is known to have neuroinvasive capacity**, causing multiple neurological symptoms with increased neuroinflammation and bloodbrain barrier (BBB) damage. The **viral spike protein disseminates via circulation during infection, and when reaching the brain could possibly cross the BBB, which was demonstrated in mice…** The aim of this study was to evaluate the barrier penetration of the S1 subunit of spike protein in model systems of human organs highly exposed to the infection. For this purpose, **in vitro human BBB and intestinal barrier cell–culture systems were investigated** by an optical biosensing method. **We found that spike protein crossed the human brain endothelial cell barrier effectively.”***

Penetration of the SARS-CoV-2 Spike Protein across the Blood–Brain Barrier, as Revealed by a

Combination of a Human Cell Culture Model System and Optical Biosensing

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8773803/

-“Our findings show that the SARS-CoV-2 spike S1 subunit protein upregulated BACE1 expression in HBMVECs [human brain microvascular endothelial cells], causing endothelial leakage. In addition, the SARS-CoV-2 spike S1 subunit protein induced p16 and p21 expression, indicating BACE1-mediated cellular senescence, confirmed by b-Gal staining in HBMVECs. In conclusion, this study demonstrated that BACE1-mediated endothelial cell damage and senescence may be linked to CVD after COVID-19 infection.”

*“In our previous study, we found ACE2 expression in endothelial cells, which increased CVD with high-risk factors for COVID-19 infection, such as diabetes and smoking. Notably, the expression of ACE2 in endothelial cells and perivascular pericytes in the brain suggests a brain-infecting potential of SARS-CoV-2. A clinical study showed that **SARS-CoV-2-infected endothelium is observed in the postmortem organs of patients with COVID-19**, which might be responsible for endothelial dysfunction. SARS-CoV-2 can directly infect human blood vessel organoids in vitro. Recently, increasing evidence has*

suggested that SARS-CoV-2 impairs brain vessels that may be linked to CVD."

SARS-CoV-2 spike S1 subunit protein-mediated increase of beta-secretase 1 (BACE1) impairs human brain vessel cells

https://pubmed.ncbi.nlm.nih.gov/35970046/

-*"pharmacokinetic studies indicate that famotidine can reach concentrations in blood that suffice to antagonize histamine H2 receptors expressed in mast cells, neutrophils, and eosinophils, these observations explain how famotidine may contribute to the reduced histamine-induced inflammation and cytokine release, thereby improving the outcome for patients with COVID-19."*

Research strongly suggests COVID-19 virus enters the brain

https://sunnylanblog.wordpress.com/2022/02/13/the-s1-spike-protein-likely-causes-thebrain-to-release-inflammatory-products-causing-a-cytokine-storm-in-the-brain/

-**New-Onset Neurologic Symptoms and Related Neuro-Oncologic Lesions Discovered After COVID-19 Vaccination:**

Two Neurosurgical Cases and Review of Post-Vaccine Inflammatory Responses

https://pubmed.ncbi.nlm.nih.gov/34277256/

-*"The pulmonary pathological findings in COVID-19 seems to be the result from the release of multiple proinflammatory cytokines, and a key source of such cytokines and chemokines is the mast cells, which are ubiquitous in the body and especially the lungs [8,9]. It has also been described that many patients who either recovered from or had mild symptoms after covid, show diffuse, multiorgan symptoms months after the infection, including malaise, myalgias, chest tightness, brain fog and other neuropsychiatric symptoms, quite similar to those presented in mast cell activation syndrome (MCAS) [8]. It has been hypothesized that COVID-19 hyperinflammation and post-COVID-19 illness may be due to a mast cell activation syndrome [3]."*

Antihistamines as an early treatment for Covid-19

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10129342/

-**Effect of famotidine [Pepcid H1-Blocker] on cognitive and behavioral dysfunctions induced in post-COVID-19 infection**

https://www.sciencedirect.com/science/article/pii/S0022399923002465

-*"Our findings suggest that SARS-CoV-2 S1 protein directly induces neuritic dystrophy, which could contribute to the high incidence of neurological disorders associated with COVID-19… The main finding of our study was that exogenous*

SARSCoV-2 S1 protein, which enters neurons via receptor-mediated endocytosis, induced endolysosome dysfunction and neurite dystrophy in neurons; such a finding provides evidence that SARS-CoV-2 S1 protein could directly induce neuronal injury."

Effect of famotidine on cognitive and behavioral dysfunctions induced in post-COVID-19 infection

https://www.frontiersin.org/articles/10.3389/fncel.2021.777738/full

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"The histamine H2 receptor targeted by famotidine is not limited to the stomach, but is also found in the brain, the endocrine and exocrine glands, the pulmonary system, and the cardiovascular system. H2 receptors are also present on mast cells (MCs), which are deregulated in viral infections including those caused by coronaviruses (8–10). Studies show that famotidine (unlike cimetidine) reaches systemic concentrations that are sufficient to antagonize H2 receptors on other cell types such as those on MCs and neutrophils (9)."

Famotidine inhibits toll-like receptor 3-mediated inflammatory signaling in SARS-CoV-2 infection

https://pubmed.ncbi.nlm.nih.gov/34214498/

-**Foodborne Viral Pathogens and Infective Protein [Prions]**

https://link.springer.com/chapter/10.1007/978-1-4939-7349-1_6

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