Below are scientific studies, which assert that SARS-CoV-2 spike protein contains a "prion-like" structure which creates abnormally folded proteins known as prions resulting in neuronal spongiosis and Parkinson, Alzheimer's type neurological disorders in the following animal studies. Prions are abnormally folded proteins that cause spongiform encephalitis with no known treatment referred to as:
Creutzfeldt-Jakob disease (CJD)-humans, Mad Cow Disease –cattle, Chronic Wasting Disease -deer

Personal research source regarding the prion issues comes from Kevin McCairn, PhD who lives in Japan and has been a neurological investigator of brain disorders 20 years mapping areas responsible for brain function. He conducts recorded live streams. Please see his video entitled: SARS-CoV-2 An Aerosolized Prion Disease - And Potential Symptomatic Profile.

From the NIH: Transmissible spongiform encephalopathies (TSEs), also known as prion diseases, are a group of rare degenerative brain disorders characterized by tiny holes that give the brain a "spongy" appearance. These holes can be seen when brain tissue is viewed under a microscope.

SARS-CoV-2 infection creates massive blood clot issues and a public awareness campaign is needed to disclose warning symptoms and appropriate preventive care may be necessary. This pandemic is increasing reports of adverse mental health conditions. It is critical that individuals who exhibit psychotic behavior be treated as a physical condition and not only regarded as a psychiatric condition. They should not presumed to be "psycho or manic" and treated with psychiatric drugs which can make the condition worse and does not address the physical root cause of brain inflammation from the viral infection.
I stand ready to assist any office with contacts to international and national researchers and physicians to discuss in more detail the evidence showing the SARS-CoV-2 is an engineered bioweapon and potential risks and to develop recommendations for best practices for public health actions and notices. Research is needed to determine the potential of this virus to produce prion replication which moves the long term concerns from a virus infection with potential to build personal immunity to an untreatable, aerosoled contageon with no treatment options.

Thank you for your review of the information.
Sheila Hemphill, CEO

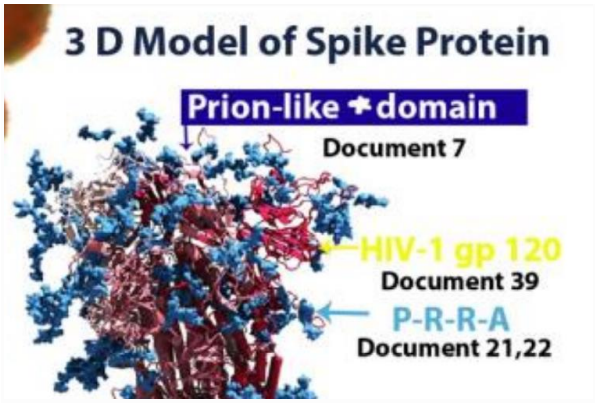
Studies indicating the prion-like structure are as follows:

1. March 29, 2020 – **Title of Study:**
   SARS-CoV-2 prion-like domains in spike proteins enable higher affinity to ACE2
   Study Conducted by:

Human Microbiology Institute, New York, NY, George Tetz and Victor Tetz.
Pre-print – this study is not peer reviewed, however same team in 2018 had published paper showing that their computer algorithms were able to detect prion-like domains in other viruses**.**

Study Highlights: *In this in silico study, using the PLAAC algorithm, we identified the presence of prion-like domains in the SARS-CoV-2 spike protein. Compared with other viruses, a striking difference was observed in the distribution of prion-like domains in the spike protein, since SARS-CoV-2 was the only coronavirus with a prion-like domain found in the receptor-binding domain of the S1 region of the spike protein.*

Note: A "prion-like domain" is not a prion – it is a region on the SARS-CoV-2 spike protein that is in the native virus or from the spike proteins created from any COVID-19 vaccine. (think jello-mold is the prion-like domain and jello is the prion which is an abnormally folded protein.)



Note: Though this report is still not peer reviewed, on March 5, 2021 Dr. Kevin McCairn, PhD a 20+ year neurological investigator on brain disorders in Japan and Dr. Richard M. Fleming, MD, PhD in Dallas, InflammoThrombic Repose expert, both who have served as peer reviewers for journals, reviewed the region and determined that the chemical attractions of the bonds in the prion-like domain (jello-mold) would create a prion.

2. On April 17, 2020, Forbes magazine published an article entitled *"Do Vampire-Like Proteins Make Coronavirus More Contagious*?"
   Highlights:
   Prions are self-replicating proteins that cause infectious brain diseases. They have misfolded structures and, like zombies that turns people into more zombies, a prion that comes into contact with its normal form will prompt that protein to adopt an abnormal shape too, triggering a chain reaction. Zombie proteins then accumulate to form aggregates of toxic fibres that kill neurones. Nearby cells clear those fibres but leave behind holes to turn tissue to sponge, as in Bovine Spongiform Encephalopathy (mad cow disease) and Creutzfeldt-Jakob

Disease in humans.

3. January 15, 2021 - **Title of Study:** Fatal neuroinvasion of SARS-CoV-2 in K18-hACE2 mice is partially dependent on2 hACE2 expression – Preprint, study is not peer reviewed.
   Study Conducted by:
   a. From Boston University School of Medicine
      Departments of:
      i. Pathology and Laboratory Medicine
      ii. Microbiology
      iii. Medicine
      iv. Biochemistry
   b. Louisiana Animal Disease Diagnostic Laboratory (LADDL) and Department of 17 Pathobiological Sciences, School of Veterinary Medicine, Louisiana State University
   c. Center for Thrombosis and Hemostasis, University Medical Center of the Johannes Gutenberg-University, Mainz, Germany

   Note: Transgenic mice have been genetically altered to possess human ACE2 receptors (door ways in the cells) on the mice cells and are used for research.

   Study Highlights:
   d. Transgenic mice expressing 40 human angiotensin-converting enzyme 2 (hACE2) under the cytokeratin 18 promoter 41 (K18-hACE2) represent a lethal model of SARS-CoV-2 infection. Pg3
   e. Despite infection and moderate inflammation in the lungs, lethality was invariably 45 associated with viral neuroinvasion and neuronal damage (including spinal motor 46 neurons). Neuroinvasion occurred following virus transport through the olfactory 47 neuroepithelium in a manner that was only partially dependent on hACE2. Pg 3
   f. The low binding affinity between the S protein and murine ACE2 (mACE2) 95 renders conventional mouse strains naturally resistant to infection, posing a challenge in 96 the development of murine models of COVID-19.
      Note: The FDA waived animal testing requirements for COVID-19 vaccine authorizations. It appears that the neurological efforts seen in the human transgenic mice are not seen in non-genetically altered, natural mice.
   g. **RESULTS:**
      i. SARS-CoV-2 is invariably fatal in infected K18-hACE2 mice with evidence of neuroinvasion.
      ii. K18-hACE2 mice inoculated intranasally with SARS-CoV-2 ($1x10^6$ plaque-forming units [PFU]; n=47 [n=25 male and n=12 female) began losing weight as early as 2-3 **days post-infection (dpi)** irrespective of sex, with maximum weight loss occurring at 7 dpi (17.7% ± 7.8% in male mice, 21.8% ± 3.1% in female, and combined 18.9% ± 6.9%; Figure 1A).
      iii. Trends in weight loss were associated with an increase in clinical scores and a decline in core body temperature, both of which became most

pronounced near the time of death (Figure 1B-C). SARS-CoV-2-infected K18-hACE2 mice exhibited neurological signs by 6-7 dpi, characterized by tremors, proprioceptive defects, abnormal gait and imbalance. **The majority of the animals were either euthanized at this time or were found dead in their cage (93%; 28/30** [Figure 1D]). Pg 4&5

iv. Altogether, these data illustrate that lethality was associated with increasing viral load in the brain until time of death, in contrast to lung viral load. Pg 6

v. Survival curve analysis clearly demonstrated that 491 lethality in infected mice only occurs after 6 dpi and in the vast majority of mice (96.3%),

vi. While the K18-hACE2 457 murine model has been critical in shedding light on mechanisms of lung injury and 458 dysfunction, it fails to faithfully recapitulate several key histologic features of severe and 459 lethal cases of COVID-19 in humans, such as diffuse alveolar damage (DAD) with 460 hyaline membrane formation and multi-organ failure associated with hypercoagulability 461 and widespread microthrombi formation (Maiese et al., 2020; Martines et al., 2020). Pg 18

vii. Our results unequivocally 507 demonstrate that neuroinvasion represents the driving component of fatality in this (which was not certified by peer review) is the author/funder. All rights reserved. No reuse allowed without permission. bioRxiv preprint doi: https://doi.org/10.1101/2021.01.13.425144; this version posted January 15, 2021. The copyright holder for this preprint 21 508 animal model compared to others such as Syrian hamsters, which display more severe 509 pulmonary disease and infection of the ONE, but no evidence of neuroinvasion. Pg 20

4. February 23, 2020 **Study Title:** SARS-CoV-2 causes brain inflammation and induces Lewy body formation in macaques

Study Conducted in the Netherlands by:

[1]*Biomedical Primate Research Centre (BPRC), Lange Kleiweg 161, 2288GJ Rijswijk*
[2]*Department of Pathology, Amsterdam UMC, De Boelelaan 1117, 1081HV Amsterdam*
[3]*Department of Biology, Theoretical Biology and Bioinformatics, Utrecht University, Padualaan 8, Utrecht*
[4]*Department Population Health Sciences, Division of Animals in Science and Society, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 1, 3584CL Utrecht*
[5]*Department of Radiology, Leiden University Medical Center, Albinusdreef 2, 2333ZA Leiden*
[6]*Biomedical Photonic Imaging Group, University of Twente, Drienerlolaan 5, 7522ND Enschede,*

One-Sentence Summar SARS-CoV-2 causes brain inflammation and Lewy bodies, a hallmark for Parkinson, after an asymptomatic infection in macaques.