Short Communication

Steven Hatfill MD

Cardiac Significance of the SARS-CoV-2 Spike Protein "*Superantigen*" Sequence in the mRNA Vaccines

As evidenced by the increased cardiac troponin levels observed in many late-stage COVID-19 patients, by March 2020 it was realized that the heart was often a target organ in SARS-CoV-2 infection.[1] The mechanisms for this cardiac damage appear diversly multifactorial involving both a myocarditis initiated by the direct viral binding to the normal ACE2 receptors on the cardiac pericytes, as well as extra-cardiac processes involving myocardial ischemia.[2]

mRNA Vaccine-Induced Cardiac Injury

New data concerning COVID-19 mRNA vaccine injury, comes from a recent report on the autopsy of two adolescent deaths shortly after the administration of a second Pfizer BioNTech COVID-19 mRNA injection (Figure 1).[3] These histopathology findings demonstrate a post-vaccine pattern of injury resembling a *Norepinephrine-mediated* stress (toxic) cardiomyopathy involving a direct injury to the myocardium[4] This has some commonalities with patients diagnosed with Takotsubo cardiomyopathy.[5]

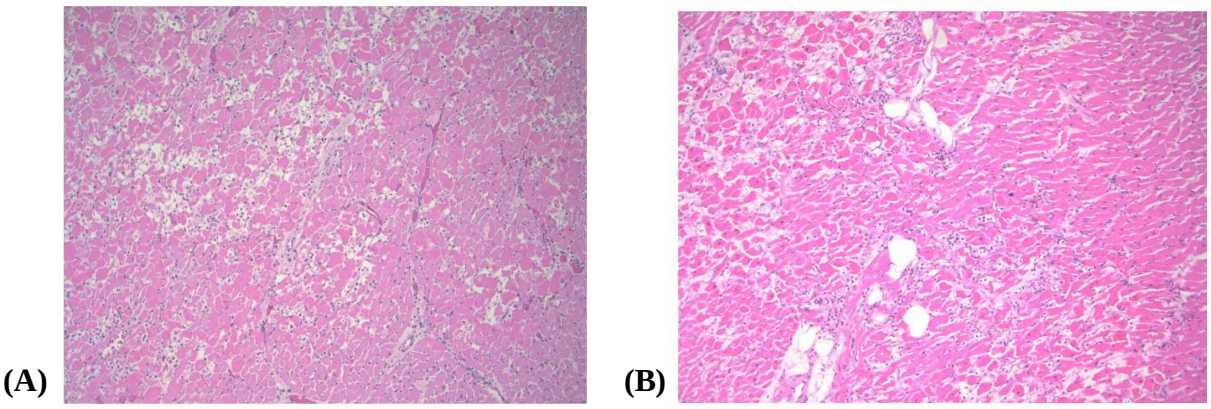

Figure 1. Case A, Heart: Confluent contraction band necrosis/coagulative myocytolysis, with a predominantly neutrophilic inflammatory infiltrate with histiocytes. H&E stain, 100X.

Case B, Heart: second sibling. Confluent areas of ischemia with coagulative myocytolysis and contraction band necrosis. H&E stain. 200X.

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Both septic shock and the hyperinflammatory side effects associated with several new cancer therapies, have revealed the existence of a self-amplifying positive feedback loop between an excess cytokine release by macrophages and the progressive abnormal elevation of circulating *Norepinephrine* levels in patients. In some instances, this expanding positive feedback loop may progress into an uncontrolled severe lethal inflammatory response called a "cytokine storm" or Cytokine Release Syndrome.[6]

The fact that cytokine storms may appear in some late-stage COVID-19 patients, prompted a search for a causative component common to both a natural SARS-CoV-2 infection and in

individuals injected with multiple doses of the COVID-19 mRNA vaccines.[7] This commonality appears to be located in a short amino acid sequence in the Spike Protein of the virus.

The mRNA Vaccines Carry a Toxin "*Superantigen*" Motif

Much has been written about the Furin Cleavage Site (FCS) of the spike protein of the SARS-CoV-2 virus. But what is largely ignored is a preceding curious stretch of ~20 amino acids (segment T678 to Q690) embedded in the S-1 monomer of the trimeric spike protein. This segment harbors a unique sequence motif that is independent from the other known SARS-related β-Coronaviruses.

In September 2020, a computational molecular modeling study revealed that this ~20 amino acid region is similar both in sequence and 3D structure to the functional motif of *Staphylococcal Enterotoxin B (SEB)* (amino acid sequence T_{150}NKKKATVQELD$_{161}$) a potent bacterial "superantigen" (SAg) toxin produced by gram-positive Staphylococcus aureus bacteria (Figure 2).[8, 9] It is of note that the SEB toxin itself is potent enough to be classified as a Category B select agent and that it was manufactured on a large-scale as a biological weapon during the 1960's.[10]

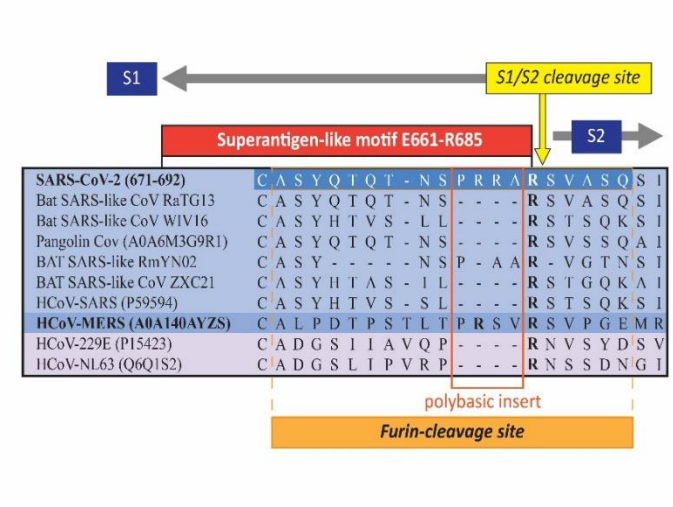

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Figure 2. Sequence alignment of SARS-CoV-2 near the S1/S2 cleavage site against multiple SARS-related strains. Viruses belonging to the same lineage are shown by the same color shade. The only other strain with furin-like cleavage site is the distantly related MERS coronavirus (highlighted in bold font). So far, the polybasic insert and the Superantigen-like motif of SARS-CoV-2 is not found in any closely related SARS-like CoV. Illustration by Brian Scaglione at the P-Value Group

Note that the PRRA insert and the furin cleavage site of the S1 subunit of the SARS-CoV-2 spike protein is at the end of a motif of ~20 amino acids *associated with a Superantigen (SAg)-like activity that can mediate a high-affinity, specific binding to T-cell receptors irrespective of their antigenic clonality.* Both structurally and functionally, the SAg-motif is closely similar the bacterial toxin Staphylococcal Enterotoxin-B (SEB). The sequence for this SAg-like motif is common to both the SARS-CoV-2 virus and the mRNA vaccines.

In humans, SEB exhibits its potent SAg activity by triggering the non-specific activation of a large fraction of the body's T-cells (up to 30%) via its high-affinity crosslinking with T-cell receptors (TCR). This gross lymphocyte activation may trigger a massive toxic release of T-cell cytokines such as interferon-gamma as well as trigger the activation of macrophages which in turn flood the blood and local tissue areas with interleukin-1, interleukin-6 and tumor necrosis factor-alpha as part of a "cytokine storm".[9]

Clinical Effects of Superantigen Exposure

SEB is one of the most potent bacterial *superantigens* known to science and it can generate severe clinical symptoms in humans at concentrations as low as 0.0004 μg/kg with death from multi-organ failure and toxic shock at 0.02 μg/kg of body weight.[11]

Severe pulmonary exposure to the purified SEB *superantigen* causes fever, chills, headache and muscle pain with reactive interstitial and later pulmonary edema.[12] This is similar to late-phase COVID-19 infection in adults who may progress into an Adult Respiratory Distress Syndrome (ARDS) and septic shock.[13]

mRNA Viral and Vaccine-Induced Superantigen Injury

The Spike Protein of SARS-CoV-2 is a trimeric transmembrane protein responsible for host cell recognition, attachment, and entry of the virus. It features two extremely large monomers termed S1 and S2 each containing roughly 1300 amino acid residues. These are fixed to a protein backbone to form the complete trimeric 3D Spike Protein structure.[14]

After intracellular viral replication, the new daughter viruses are transported back to the surface of the cell where they are released to infect more normal cells. As part of this process the virus's S1 monomer is cleaved at the Furin cleavage site and it changes its 3D conformation from a closed into an open configuration (Figure 3).

This now open conformation exposes the ACE-2 Receptor Binding Domain (RBD) of the S1 monomer which can now fully recognize the ACE Receptors on the surface of uninfected cells (Figure 3). At the same time, the amino acid sequences forming the SEB SAg-like motif are also now exposed to the outside where they are accessible for T-Cell Receptor binding to cause large-scale polyclonal T-cell activation and proliferation. [14]

As previously mentioned, the bacterial SEB SAg toxin exerts its superantigen effects by inducing an inflammatory cytokine release from T-cells and macrophages.. Of note is that this SEB-released cytokine profile is similar to the cytokine profile in COVID-19 patients with a prognosis of severe infection and death.[15] This includes elevated levels of IL-6, TNFα, IL-8 and IL-1β which leads to multiorgan tissue damage.

This is also observed in infected children that develop COVID-19 associated Multisystem Inflammatory Syndrome (MIS-C). [16, 17] An abnormal cytokine profile is also a feature of COVID-19 "Long Hauler Syndrome".[18] A cohort analysis of adult COVID-19 patients reveals that cases with severe hyperinflammatory disease also exhibit a marked skewing of the TCR repertoire that is consistent with *superantigen* activity.[8] Another study indicates that the rare SARS-CoV-2 Spike Protein mutation (D839Y/N/E) detected in a European strain of the COVID virus, may potentially further enhance TCR binding in late-phase COVID-19 patients.[19]

It is therefore reasonable to assume that the hyperinflammatory syndrome of COVID-19 originates from the superantigen sequence embedded into the S1 monomer of the spike protein in both SARS-CoV-2 and the mRNA vaccines.

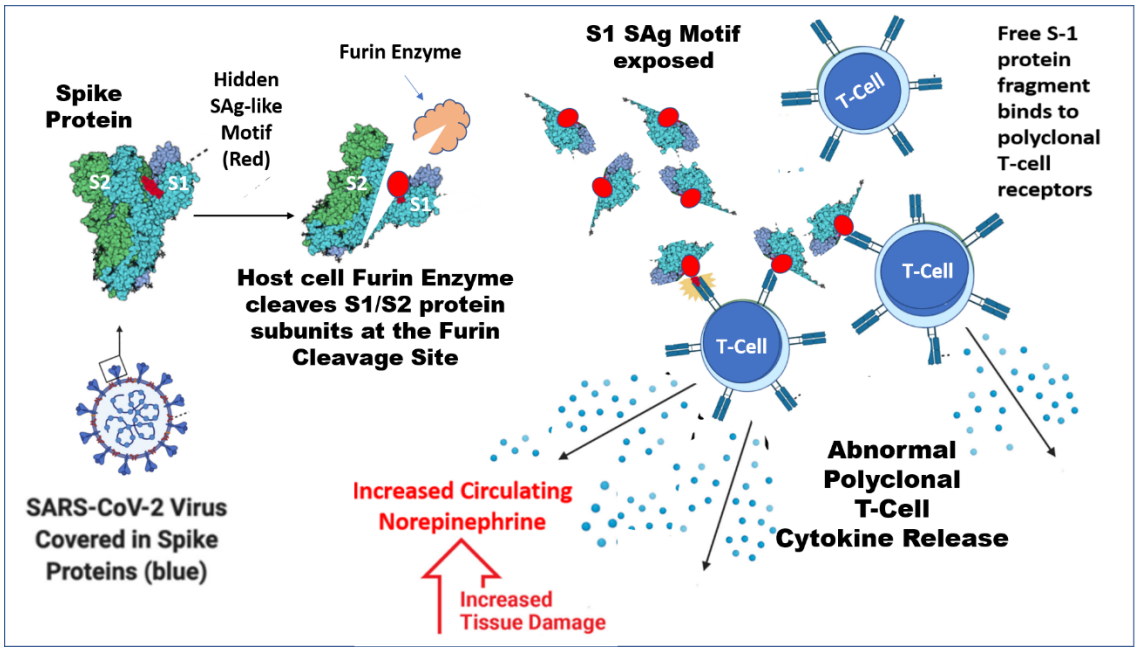

Figure 3. Left Images: *When new daughter viruses are transported back to the surface of the infected cell, the virus's S1 monomer is cleaved at the Furin cleavage site. This changes its 3D conformation from a normally "closed" into an "open" configuration. This open configuration fully exposes the SEB Sag-like motif of cleaved S1 subunit of the SARS-CoV-2 virus to the exterior of the host cell membrane. The sequence for this superantigen motif is found in both the SARS-CoV-2 virus and the mRNA vaccines.* Illustration by Brian Scaglione at the P-Value Group

Right Images: *The exposed* SAg-like motif *is free to recognize and bind to the T-cell receptors found on circulating cytotoxic T-cell lymphocytes. This leads to a massive production of circulating proinflammatory cytokines including IFNγ, TNFα, and IL-2 from T cells, as well as IL-1 and TNFα from macrophages. This is theorized to cause a positive feedback loop between elevated cytokine levels and circulating Norepinephrine levels leading to severe cardiac damage.*

Based on the above data, it is reasonable to propose that in some cases, the *superantigen* motif in the Spike Protein of SARS-CoV-2, can trigger an uncontrolled positive feedback loop between excessive cytokine release and excessive *Norepinephrine* release causing both cytotoxic and ischemic myocardial damage. This is expected to occur in natural COVID-19 infections and in some mRNA vaccine recipients, as well as in rare cases of the Multisystem Inflammatory Syndrome (MIS-C) observed in COVID-19-infected European and American children.[3, 17]

More Urgent Studies Needed

There is an express need to acquire more data on *Norepinephrine*-induced myocardial injury in mRNA vaccine recipients. These studies should remain focused on new immunizations involving the original mRNA vaccines.

The new studies should not include Omicron and its subvariant clinical cases, or the recipients of the new Omicron mRNA vaccine. This is because of the extensive mutational changes that have occurred in the Spike Protein of the Omicron clade.[20] Omicron exhibits mutations in the P681RRAR685 putative SAg-like segment of the S1 subunit with possible changes in Spike Protein cleavage and exposure of the C-terminal end R681RRAR685 of the superantigenic segment accompanied by changes in viral fitness, infectivity, and apparently a lower virulence.[21]

The clinical studies on new recipients of the traditional Moderna and Pfizer vaccines should include weekly flow-cytometry assessments and measurements of circulating catecholamine levels, particularly Norepinephrine. Existing cardiac autopsy material could also be re-examined and partially characterized with immunohistochemical studies.

Clinical research should also be conducted with respect to the early clinical use of β2-receptor antagonists to diminish any developing positive feedback loop between catecholamine-driven signaling and hyperinflammation in both active COVID-19 infection, long-hauler syndrome, and non-Omicron post-vaccine injuries.[22]

Other Food and Drug Administration-approved anti-inflammatory drugs such as the antibiotic Minocycline[23] and the inexpensive drug Indomethacin (which has shown value in SARS-1 infection),[24] should be combinational-tested in animal models of SEB-induced Toxic Shock Syndrome in addition to CTLA4-Ig which can inhibit CD28 co-stimulation of cytotoxic T-cells,[25] and the mTOR inhibitor Rapamycin.[26]

As a final note, the analogue *superantigen* sequence embedded into the virus lacks any defined viral evolutionary lineage. To the best of knowledge, no other SARS-related β-Coronaviruses outside of SARS-CoV-2, has this SEB-analogue sequence. As previously discussed, the SEB toxin is classified as a Category B select agent and it was manufactured on a large-scale as a biological weapon during the 1960's.[27]

Lacking a defined natural evolutionary lineage for the SEB-like motif in the S1 monomer, a consideration must be given for a laboratory origin for SARS-CoV-2. In this respect the Wuhan Institute of Virology is often mentioned. However, it must be remembered that the presence of the SAg-like motif is covert and is not detected by normal protein or nucleotide BLAST Search (*Basic Local Alignment* Search *Tool*) efforts. It was only discovered after a structure and dynamics analysis.

The creation of the mRNA for this S1 SEB SAg-like sequence would have required full-atomic structure and dynamics analyses necessitating the cooperation of several different large, well-funded, Chinese laboratories. This would include an experienced proteomics facility with specialized equipment and expertise not generally available in a dedicated virology laboratory.

It is also of note that the former Soviet Biopreparat offensive biological weapons program was experimenting with the genetic insertion of peptide toxins and immunogenetic peptides (Myelin Basic Protein) into known biological warfare pathogens during the 1990s.[28, 29] Also that previous statements allegedly made in the 2003 time-frame by the Chinese Defense Minister Chi Haotian have been reported to advocate the Chinese continuation of this type of *Gain-of-Function* Soviet offensive biological warfare research.[30]

Although the majority of SARS-CoV-2 infections have been accompanied with a very low mortality rate, if the SEB Sag-like motif present in the virus proves to be an intentional insert, this technology represents a dramatic advancement in offensive biological warfare using incapacitating agents and as has now been witnessed, a direct severe strategic threat to the United States and its allies.

America Deserves Answers

The discovery of the SEB-like functional superantigen motif in the Spike Protein of SARS-CoV-2 was published as an Open Access report in the Proceedings of the National Academy of Sciences on 28 September 2020. Yet only now is the clinical significance of the *SAg-like* motif in the spike protein of the virus receiving attention and debate.

This is inexcusable considering millions of Americans and Europeans have already been injected with this mRNA sequence as part of the global mRNA vaccination program. This is a pseudo-mRNA injection that can remain in the body for months.

When published in 2020, the *in-silico* computational modelling report should have triggered an immediate vaccine review by Dr. Anthony Fauci and the COVID-19 Task Force, Dr. Francis Collins at the NIH, Professor Arnold Monto at the University of Michigan (the Chair of the outside panelists at the FDA Center for Biologics Evaluation), and the FDA Commissioner Dr. Stephen Hahn together with Dr. Janet Woodcock, and Dr. Peter Marks. It also should have included Moncef Slaoui, the PhD-level scientist who was tasked to manage the Operation Warp Speed project as well as representatives from the vaccine manufacturers.

To the best of knowledge, no such meeting concerning the COVID-19 superantigen motif ever occurred, yet questions over mRNA vaccine safety had appeared as early as February of 2021. By March 2021, more than 20 European countries had temporarily stopped using AstraZeneca's COVID-19 vaccine for a safety review. In April 2021, a short temporary 11-day hold was placed on the Johnson and Johnson mRNA vaccine The extensive serious adverse events associated with the Moderna and Pfizer vaccines are currently the subject of ongoing discussion with many countries now moving away from their ineffective mRNA vaccination mandates.

Throughout 2021 and into 2022, the number of recorded serious, adverse, post-injection clinical events has dramatically intensified with the conscious effort to apply duress to Americans hesitant to receive the still highly experimental non-FDA approved mRNA vaccines. This is in addition to the intense promotion of clinically unnecessary childhood vaccination using these preparations.

It is of note that the average number of deaths attributed to the widespread use of the annual influenza vaccine is 39 with roughly 43% of the US population vaccinated.[31]

The average number of deaths attributed to the mRNA vaccinations for COVID-19 is a shocking estimated 20,622 with roughly 65.8% of the total population vaccinated. Many of these mRNA vaccine-associated deaths are accompanied by bizarre clotting phenomena.[32, 33]

America deserves clear answers for the decisions and lack of honesty by federal, state, and local authorities during the COVID -19 pandemic response. Most particularly concerning the intentional block of cheap effective outpatient anti-viral drugs and the coerced use of poorly effective, experimental, mRNA vaccines. Individual accountability must be assigned.

Acknowledgements

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Author

Dr. Steven Hatfill is a medical doctor a virologist, and a Senior Fellow at the London Center for Policy Analysis. His Medical Fellowships include the NIH, Oxford University, and USAMRIID where he studied the Ebola and Monkeypox viruses. He is the author of *"Three Seconds Until Midnight"* prophetically published two months before the US COVID-19 outbreak. He is the scientific author for *"The CCP is at War With America,"* and has numerous peer-reviewed scientific publications in virology and hematological pathology. From 3 February 2020 until the 2021 transition, he served as a senior medical advisor for COVID-19 to the Executive Office of the President of the United States.

Dr. Hatfill has no financial conflicts of interest with vaccines or with outpatient treatments for COVID-19.

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