INACTIVATION OF COVID-19 VIA ANTIVIRAL MACROMOLECULES

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WHAT MAKES COVID-19 SO HARD TO DEFEAT

COVID-19 is difficult to render biologically inactive using the common single point of failure methodology employed to address a typical influenza virus. CoVID-19 has proven more akin to HIV in that it has a retroviral component in addition to a lipid membrane with multiple surface-binding proteins exhibiting enhanced ACE2 receptor affinity. (1,2)

HOW WE CAN FIGHT IT?

A new tactic has been employed to exploit multiple weaknesses in its replicative and proliferative processes during both the asymptomatic and acute phases.

PHASE 1 OF THE ASSAULT

Get through the cell membrane of COVID-19 - A novel macromolecule was designed using a Carbon cage adducted to a lipid, thus allowing it clear passage through cell membranes, (3,4)

Deliver the Payload That Will Kill COVID-19 - Serum with a fat-soluble synthetic antioxidant, dibutylhydroxytoluene, and 3,3',4',5,7-Pentahydroxyflavone as molecular addends is delivered once the cell membrane is cleared.(5)

Protect the Payload so it can do its job -Lastly, this serum is then emulsified with a proteolytic enzyme, whereby it protects the enzymatic payload.(6)

PHASE 2 OF THE ASSAULT

Relieve acute phase of the respiratory syndrome - The enzymes are then delivered to the lungs where they consume sclerotic tissue and relieve the fibrinolytic burden placed on the alveoli during the acute phase of the respiratory syndrome. (7,8)

Destroy Active COVID-19 - 3,3',4',5,7-Pentahydroxyflavone expresses a high affinity for S-protein:ACE2 ligand interaction, thereby creating interference and inhibiting virion/host cell interaction. BHT (dibutylhydroxytoluene) is employed to destroy the active CoVid-19 viruses by breaking down the lipid membrane that encapsulates them (9,10) Similarly, the carbon derivatives will inhibit the retroviral component of the virus by binding to the SARS-CoV-19 MPRO, thus blocking conversion to an activated virion via Main protease (MPRO) inhibition. Previous research has conclusively proven using HIV viral strains and ELISA testing that myriad effects are possible based on the various moieties and macro-molecule configurations to disrupt protease binding. (11,12)

History of This Approach - During the internal development process of the original antiviral macromolecule from which this therapeutic evolved, we were able to eliminate other lipid enveloped viruses both in vitro and in vivo. To date we have eliminated HSV-1 and Epstein Barr. Participants were re-assessed after treatment, and no active antibodies were present. **The base formula has also been shown to be effective against HTLV-1, the AIDS virus, in a published, peer-reviewed journal.** This is however not evidentiary data sufficient to reach a conclusion of full viral destruction, but as all patients became asymptomatic and continued to remain so, it does provide anecdotal support for rapid viral mitigation and symptom relief. We are in process with the ongoing development and plan to file an IND with the FDA in 2021. However, due to the pressing nature of the current viral threat, we feel that bringing this research to light is incumbent upon us.

Rapid Relief of Acute Respiratory Distress is Likely - This multi-pronged approach allows for an immediate suppression of cytokines which both inhibit viral replication and eliminate the possibility of a cytokine storm. During the initial two-hour period post administration while the cytokines are being dropped, serratiopeptidase (a proteolytic enzyme Serratia E-15 derivative) in the serum begins rapidly digesting the fibrinolytic buildups caused by the virus. This allows for rapid relief of acute respiratory distress and lessens the likelihood of negative patient outcomes.

The components exhibit a low toxicity profile and many are currently categorized as GRAS by the FDA.

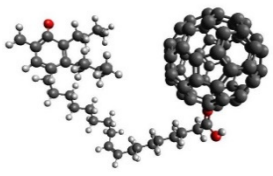

Figure 1. Antiviral Macromolecule – Serves as a pseudo-vaccine in that it forces asymptomatic biologic activity while a person is exposed to the virus so that their own innate immune system can develop antibodies to the virus. The Fullerene-mediated suppression of cytokines mitigates the sclerotic lung damage that is currently being seen even in healthy patients post infection.

References

1 - Ben M Dunn, M. M. (2002, March). Protein family review Retroviral proteases. Genome Biology, 3(4). Retrieved from http://genomebiology.com/2002/3/4/reviews/3006.3

2 - Young, E. (2020, March). Why the Coronavirus Has Been So Successful. The Atlantic. Retrieved from https://www.theatlantic.com/science/archive/2020/03/biography-new-coronavirus/608338/

3 - Baati, T. et al. (2012): "The prolongation of the lifespan of rats by repeated oral administration of [60] Fullerene.", BIOMATERIALS, pages 1 - 11 *

4 - Mitchell, Ian et al. (2014): Cellular enhancements in biological systems through the use of lipofullerene and peptide group combinations, United States Patent US9308243B2

5 - Branen, A. (1975, February 01). Toxicology and Biochemistry of butylated hydroxyanisole and butylated hydroxytoluene. Journal of the American Oil Chemists Society, 52(2). doi:10.1007/BF02901825

6 - Craven, J.F. (1953) Treatment of obstructive atelectasis by aerosol administration of proteolytic enzymes. The Journal of Pediatrics, Volume 42, Issue 2, pages 228 - 230

7 - Kumar, S. et al. (2013) "Preparation, characterization and targeted delivery of serratiopeptidase immobilized on amino-functionalized magnetic nanoparticles.", EUROPEAN J PHARM BIOPHARM., vol. 85 (3 Pt, 10 July 2013, pages 413 - 26

8 - White, E.S., Lazar, M.H. and Thannickal, V.J. (2003), Pathogenetic mechanisms in usual interstitial pneumonia/idiopathic pulmonary fibrosis. J. Pathol., 201: 343-354. doi:10.1002/path.1446

9 - K.S. Kim, H. M. (1978, July). Inactivation of Cytomegalovirus and Semliki Forest Virus by Butylated Hydroxytoluene. The Journal of Infectious Diseases, 188(1), 91-94. Retrieved from http://jid.oxfordjournals.org/

10 - W Snipes, S. P. (1975). Butylated hydroxytoluene inactivated lipid-containing viruses. Science, 188, 64-66. doi:10.1126/science.163494

11 - Simon H. Friedman, Padma S. Ganapathi, Yves Rubin, and George L. Kenyon, (1998) “Optimizing the Binding of Fullerene Inhibitors of the HIV-1 Protease through Predicted Increases in Hydrophobic Desolvation”, Journal of Medicinal Chemistry, 41 (13), 2424-2429 DOI: 10.1021/jm970689r

12 - Zhongwei Zhu, David I. Schuster, and Mark E. Tuckerman. (2003) “Molecular Dynamics Study of the Connection between Flap Closing and Binding of Fullerene-Based Inhibitors of the HIV-1 Protease.” Biochemistry, 42 (5), 1326-1333. DOI: 10.1021/bi020496s.