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How to cripple SARS-COV-2 virus with Ozone treatment

Esin Eren¹ , Ferhat Saribek², Zafer Kalayci³,Necat Yilmaz¹

¹ Department of Clinical Chemistry and LC/MS-MS Laboratory, Antalya Training and Research Hospital, Antalya, Turkey

² Ozone Therapy Unit ,Kepez State Hospital ,Antalya, Turkey.

³ Traditional and Complementary Medicine Head of Department of Ministry of Health Ankara Turkey

Thiol groups in viruses and SARS-COV-2

Corona virus and other viruses fortunately have no self-repair mechanism, unlike “living” cells. Cysteine is an amino acid carrying a sulfhydryl (R-S-H) residue, also called a “thiol” group and the S-H functional group is called the thiol group or the sulfhydryl group. In order to successfully penetrate cells, many viruses (e.g. SARS-COV-2) require membrane glycoproteins to be in reduced S-H form rather than oxidized (R-S-S-R). For example HIV is dependent on reduced sulfhydryl groups for infectivity, as also reported for Ebola virus to enter cells. Like Ebola, corona virus structure also has regions rich in cysteine, inclusive of the spike and envelope proteins(1-4).

(S) Protein and cysteine amino acids and cripple virus

We would now like to draw attention to the main important mechanism, the host cell receptor of active Cysteine viruses (e.g. angiotensin converting enzyme receptor) is essential for membrane fusion! Undoubtedly the redox status (reduced cysteine residues vs. oxidized residues) may be “switch” protein activity to “on” or “off for SARS-COV-2. Alterations of these sulfhydryl residues have been found to “Cripple” virus growth properties at least two logs lower than wild type virus. In fact Cysteine is highly vulnerable to oxidation to disulfide (R-S-S-R) or other residues; which effect will cripple its biochemical activity in proteins, altering their three-dimensional structure (1,5-8).

SARS-COV-2 relationship between atmospheric oxygen and temperature

Simply atmospheric oxygen may slowly degrade these sulfhydryl (thiol) groups in viruses, and do so more quickly at higher temperatures these cripple effect on viruses! Indeed thiol S-H bonds are far weaker than the O-H bonds in alcohols, and vulnerable to oxygen based oxidants, which can oxidize the sulfur to sulfonic acid residues (R-SO₃-H). So Corona viruses

have a limited “shelf life” on surfaces. SARS-COV-2 reportedly retain infectivity up to one week on surfaces, temperature dependent, and are quickly inactivated by oxidizing disinfectants (3, 8).

Ozone treatment may be ideal therapy for viruses

Ozone is triatomic oxygen (O₃), the most powerful oxidant found in nature. Our bodies actually produce ozone against many infectious agents. Because Ozone inactivates many viruses directly. The information that now forms the basic hypothesis of this paper is that, OZONE it will oxidize thiol (S-H) groups at the moment of contact! Creating a more “oxidized” environment, ozone therapy may assist the body in inactivating thiols in viruses in blood and tissues. Furthermore, SARS-COV-2 spike protein(S) is also rich in Tryptophan amino acids, which is second to Cysteine in vulnerability to oxidation. Because knowing ozone extinguishes itself virtually instantly on contact with blood, creating “Ozonides”. Ozonides are oxidants in their own right. When blood is treated with ozone, it instantly reacts with electron-rich double bonds of lipids and other molecules. This creates longer lasting downstream weaker oxidant metabolites called ozonides: reactive oxygen species and lipid oxidation products, inclusive of peroxides, peroxyols, alkenes, alkanes For example, Our immune system is well known to create reactive oxidant species, such as hydrogen peroxide, superoxide, nitric oxide, hypochlorous acid, etc. and even ozone itself as to defend against infection. These molecules appear to act as messengers for the key biochemical and immune modulating effects of the therapy Ozone has a most inexpensive, safe, and likely effective remedy for deadly viral diseases, which exploits their redox vulnerability at critical membrane *cysteine/tryptophan* fusion sites(3,4,5).

Ozonides may suppressing SARS-COV-2 and cytokine storm

These ozonides molecules are less reactive than ozone, but still possess oxidizing power and serve as biochemical signaling molecules modulating the immune system. This could be exceptionally valuable as a means of safely suppressing “cytokine storm”, often the cause of final lethality from pulmonary viral infection, including coronavirus. Ozone’s ability to inactivate cysteine dependent proteins was reported as an ozonide attack on cysteine-dependent many enzyme like papain, believed to inactivate the enzyme by oxidizing the active sulfhydryl group to sulfenate or sulfenic acid(5,7).

Conclusion

Ozone therapy could be easily apply even in very poor countries. Milder cases could also be treated to study the ability of ozone therapy to slow or halt clinical deterioration. Such study could bring ozone therapy to the forefront of all-around infectious disease management, providing answers to our growing problems with viral infection (7,8).

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