

## **Protection Against Oxidative Stress as an Important Therapeutic Factor in Pandemic Coronavirus Infection**

**Václav Holecek**

Mulac Hospital, Czech Republic, Pilsen.

**Rokyta R.**

Charles University Prague, Dept of Pathophysiology

**Vlasak R.**

Prevence 2000, Prague

Oxidative stress occurs in the body of the virus host (6), the predominance of free radicals over antioxidants. Unfortunately, there is a lack of information on which and how many free radicals stress causes. Free radicals oxidatively damage macromolecules such as lipids, proteins, nucleic acids, carbohydrates (5), thus the virus obtains energy for life and rapid reproduction. Oxidation of nitrogenous bases by free radicals causes mutations of the virus, which thus resists the effect of the vaccines developed for it. Free radicals differ in half-life from 10<sup>-9</sup> seconds to an order of magnitude longer. Among other things, they accumulate in the brain during the day, and are removed in the dark during sleep. Sleep acts as an antioxidant. The combination of free radicals "free radical storm" with cytokine storm is especially dangerous. The lungs are very vulnerable to free radicals. They cannot synthesize cysteine and therefore usually lack glutathione. There is also a lack of vitamin D3 (1) and other antioxidants. Lipoperoxidation produces, among other things, malondialdehyde (MDA), whose aldehyde groups bind to free amino groups of proteins (e.g. immunoglobulins or spike proteins of vaccines) and the resulting complex loses its antiviral activity. Even a person prevents the decline of immunity by trying to reduce the level of MDA and increase the immune capacity by producing anti-MDA. MDA is also produced by the cyclooxygenase reaction on membrane lipids. Until we prevent the mutations of the virus, it will not be eradicated. Mutations can be prevented by administering small molecules of selenite or iodide, which get close to nucleic acids and are preferentially oxidized over nitrogenous bases:  $\text{Se}^{4+} - 2\text{e}^- \rightarrow \text{Se}^{6+}$  or  $2\text{I}^- - 2\text{e}^- = \text{I}_2$ . (4). An important compound is acetyl-CoA. The reaction of other acetyl-CoA molecules produces fatty acids, cholesterol and ubiquinol Q10. Other acetyl-CoA molecules can be created by the effect of sirtuins (mainly histone deacetylases, the Sars-CoV-2 virus also contains them). Sirtuins (SIRT1 – SIRT7) have an anti-aging effect and are contained in all cells, including Sars-CoV-2 (7). Blocking lipid synthesis with fenofibrate has proven effective for Israeli scientists (3). Of course, effective antioxidants with a low redox potential can be an alternative to prevent lipoperoxidation (e.g. NMN, NADPH, hydrogen, reduced glutathione (GSH)).

After a certain time, proteins "wear out", including immune antibodies, and the body has to make new ones. Old senescent cells still take nutrients and oxygen and do nothing for the body. Senolytics such as fisetin, quercetin etc. remove senescent cells.

At the age of 80, the total antioxidant capacity drops to about 10% of the original value, and therefore seniors are more at risk of this infection. (8) Free radicals also damage the protein receptors for smell and taste.

A large number of immune substances are produced in the intestine. As they pass through the intestine, they are absorbed together with minerals and other necessary substances, but free radicals, on the contrary, are concentrated and it can be assumed that this can also be the cause of colon and rectal cancers. Regular oral administration of antioxidants could reduce the risk of these cancers. (2.)

Oxidative stress is also manifested in other viral diseases, e.g. herpes, hepatitis, HIV, etc.

### **CONCLUSION**

As an additional therapy against the coronavirus, it is necessary to consider reducing oxidative stress, reducing the formation of malondialdehyde and lipids and thus lipoperoxidation, and preventing virus mutations.

### **References**

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