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CASE -REPORTS

# Possibilities of intervention in the sars-cov-2 metabolism.

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Abstract

In addition to epidemiological resources and vaccines, the virus's defenses need to be compromised in order to weaken the coronavirus pandemic. These include oxidative stress, mutations, and processing of acetyl-CoA for lipid synthesis instead of glycolysis. Antioxidants with low redox potential, selenium or iodides and fenofibrate can be used for this, which reduces the formation of malondialdehyde from lipoperoxidation. Theoretically malondialdehyde can bind to the amino group of proteins (immunoglobulins or vaccine protein) and thus protect the virus.



Th SARS-CoV-3 virus is not interested in killing its hosts, because it endangers its own life. Therefore, there are less lethal variants of the virus, but more infectious. Eradication of the virus can occur when we prevent its mutations or when the recurrence of antibodies gets into the hereditary equipment of human DNA through an epigenetic process. Experience with the pandemic infection so far shows that vaccination and epidemiological measures are not enough to completely control the infection, and therefore it is necessary to find other treatment options.

## INTREVENTIONS AGAINST SARS-COV-2 AND DISCUSSION

There are some actions damaging its defense responses. Above all, there are two characteristics:

1. SARS-CoV-2 produces oxidative stress (imbalance between free radicals and antioxidants in favor of radicals.)(2). By oxidative stress, the virus gains energy and by oxidizing nitrogen bases it creates mutations for which the organism does not yet have antibodies. A mixture of antioxidants can neutralize free radicals (4).
2. The virus redirects glucose metabolism to lipid biosyntheses (1).

Ad 1) Free radicals oxidize sugars, fats, proteins and nucleic acids. By oxidation, they gain energy resources for their lives, and in addition, the oxidation of nitrogen bases causes mutations in the virus, thereby preventing the virus from forming antibodies and reacting to the proteins of the virus itself (3). Mutations may already occur in a person who has become ill. A certain part of the virus can

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avoid the effect of antibodies by mutation and can settle somewhere in the body as a latent infection, which waits for suitable conditions for replication. This would explain some of the subsequent problems of patients who have already been "cured." We know NAD + - sirtuins (deacetylases), which are present in all cells, including viral ones, can also increase the amount of acetyl-KoA. Therefore, fenofibrate has a virucidal effect by inhibiting lipogenesis. Lipoperoxidation is inhibited by antioxidants,that selenium counteracts mutations because it has a small molecule that comes close to RNA (DNA) and can protect nitrogen bases from oxidation by preferentially oxidizing itself (Se4+-2 e- → Se6+). Iodide can work similarly: 2 I- - 2 e- = I2. (Oxidation is the loss of electrons.)

Ad 2) In its defense, the virus redirects acetyl-KoA from the TCA cycle to the synthesis of fatty acids, which it then lipoperoxidizes with free radicals to form malondialdehyde, which with its aldehyde groups can bind to the –NH2 groups of antibody proteins (Ig) and possibly the vaccine protein and thus destroy them. NAD+ - sirtuins (deacetylases), which are present in all cells, including viral ones, can also increase the amount of acetyl-KoA. Therefore, fenofibrate has a virucidal effect by inhibiting lipogenesis. Lipoperoxidation is inhibited by antioxidants.

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