Brief Communication

COVID-19 Solution

Abstract

Coronaviruses (CoV) are RNA viruses that cause endemic infections in various species of mammals and avian birds. There are seven known human CoVs, each of which causes respiratory diseases: together account for about one third of common colds. Some CoVs have recently entered humans from infected animals and lastly we have SARS COVID-19, (CoV), which causes severe acute, often fatal respiratory syndromes. The prevalence of CoV, the easy zoonotic transmission and the potential to cause serious respiratory diseases, lead to urgent research to discover the mechanisms of CoV infection. Our study has identified a possible way to eliminate the danger of this virus by analyzing the structures by which it enters the host cell. This study indicates that the neuroaminidase interrupts the infection.

Keywords: *Neuroaminidase, SARS COVID-19, sialic acid*

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Considerations and Solutions

All researchers agree that the structures that are responsable of mechanisms by which CoV-19 enters host cells and subsequently spreads, may explain, the significant expansion of these viruses into new hosts. Entry and spread are carried out by homotrimetric and by multi-domains integral membrane with a needle (S) glycoproteins structure, protruding from virions and infected cells.[1] The S proteins infect the cells by the receptors of host cells and subsequently act as catalysts for the fusion of the virus cell membrane as showed in different contexts of CoV infection, whereas clear the fusion with the cell membrane. With regard to the adhesion phase, it should be noted that many CoV S proteins contain several distinct domains that bind the host cell receptor (RBD). The presence of more than one RBD raises the question of whether the RBDs operate independently and, if so, whether one RBD provides complete expertise in infection, while the other remains as an inactive, that is, vestigial domain.

The MERS-CoV S proteins contain an N-terminal RBD (also known as the "S1A" domain).[2] Our study has allowed us to verify how this type of protein has a configuration that is structurally similar to glycoproteins of the "MUCINS" type with the final part consisting of saccharide groups, properly sialic acid that binds to the receptors of the host cells.[3]

The COVID-19 virus has a high diffusive capacity because its structure has several groups of terminal sialic acid. Our hypothesis is that this type of structure strengthens the capacity of diffusion and also depends on the number of glycoproteins present on the COVID-19 capsule. In any case, the most important parameter is the fact that this glycoproteins is similar to the salivary mucins, with structures that allow an easy attack to the host cells. In addition, the number of those structures are about double those of a normal influenza virus and in fact the RNA of COVID-19 is the longest of all other viruses. The affinity of these structures with ACE 2 receptors is similar to other viruses, and works thanks to the terminal saccharide part of the external glycoprotein neuroaminidase type, present in COVID 19.

These glycoprotein enzymes contain in their terminal structure, oligosaccharide compounds, mainly sialic acid that are necessary to infect cells Our intuition was to think that using the neuroaminidase enzyme can sever the binding of sialic acid with the rest of the glycoprotein making it impossible to link the virus to the host cell.

It's very important to note, also to understand the various pathological expressions of the virus in patients, as

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THE ENDOGENEAL PRODUCTION OF SIALINIDASIS DECREASES in various physiological and/or pathological conditions.

In fact NEUROAMINIDASI is an enzyme that our body secretes in quantities that decrease with age and this could explain why children very rarely get infected. Moreover, it decreases strongly in diabetics and hypertension, and this fact also coincides with the high number of these patients suffering from covid-19.

We have done and repeated some experiments that allowed us to verify how the use of the neuraminidase enzyme inactivated the entire structure. To verify this, we dosed the concentration of sialic acid before and after the addition of the enzyme in some nasal viruses.

Reagents

SialEXO®

The SialEXO family are sialidase products for the removal and analysis of sialic acids. The enzymes are active on N- or O-linked glycans present on native glycoproteins or released glycan structures.

SialEXO - sialidase mix for complete removal of sialic acids (α2-3, α2-6 and α2-8).

These preliminary tests were conducted on mucus secreted by the nose. After addition of neuroaminidase, the amounts of free sialic acid have increased by 90%: this means that the enzyme has broken the bonds between sugars and proteins, thus inactivating the virus' diffusive capacity.

The other important result is that free SIALINIDASI can compete with ACE2 receptors and further inhibit the binding of the virus with host cells.

The addition of the enzyme in fact causes an increase in free sialic acid, for the lysis of bridges connecting the protein structure with the oligosaccharide structure. We confirmed these results after the addition of neoroaminidase in two samples of COVID-19 extracted by water solution. We believe that the use *in vitro* of this enzyme has the same effect on COVID-19 *in vivo*.

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Conflicts of interest

There are no conflicts of interest.

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