

Neuropilin-1 in Transmission Process of COVID-19

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Letter to the Editor

What we know well so far is that SARS-CoV-2 docks to the angiotensin-2 receptor in the lungs. Recent publications by two research teams from Munich (Germany) and Bristol (Great Britain) shed light on the puzzling role of neuropilin-1 in the transmission process of COVID-19 disease. The neuropilin gene is located on the chromosome locus 10p11.22. This is one of two human neuropilins. NRP1 is a membrane-bound co-receptor to a tyrosine kinase receptor for both members of the vascular endothelial growth factor family (VEGF) and members of the semaphorin family (SEMA3a). NRP1 plays a versatile role in angiogenesis, axon control, cell survival, migration and invasion. In vivo mouse studies have shown that injection of NRP-1 inhibits the progression of acute myeloid leukemia in mice.

It has long been known that SARS-CoV-2 uses the spike protein to bind to the ACE2 receptor, which is important for the virus to enter the cell. Two papers published on a preprint server now also bring the protein neuropilin-1 (NRP1) into play, to which they also attribute a specific function in the infection of a host cell. The team of authors led by Dr. Ludovico Cantuti-Castelvetri from the University of Munich, the second by a team of scientists led by James L. Daly from the University of Bristol. Both research teams found similar results that neuropilin 1 could play an important role

in the COVID-19 transmission process. In SARS-CoV-2 infected cells, a precursor protein of the spike protein is cut open using the protease furin. This creates an amino acid sequence that enables the spike protein to bind to NRP1. It is widespread in the body and occurs primarily on cells in the lungs and nose. It is usually the binding site of the body's proteins. The fact that the spike protein can anchor itself here is an unfortunate but understandable condition. The Munich team was able to show that the SARS-CoV-2 coronavirus could significantly increase its infectivity by

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binding to the NRP1. In the nose tissue samples from Covid 19 patients, it was primarily the cells that showed the protein on their surface that were infected with the virus. It is very likely that the main entry portal for the virus into the cell is the ACE-2 receptor, but Drosten suggests that individual viruses could enter the cell if only NRP1 was present. The doctor believes that this alone would not make the virus an epidemic. But: "This additional availability of neuropilin, especially on the mucous membranes of the upper respiratory tract, could have been the decisive change in the way in which SARS-CoV-2 achieved this transferability via the upper respiratory tract and thus ultimately became a pandemic agent. The virologist also points to a difference from the old SARS pathogen, SARS-CoV-1 does not have this

furin cleavage site, Cantuti-Castelvetri and his colleagues believe that the olfactory epithelial cells express NRP1 particularly strongly and that these cells are particularly useful when examining tissue samples.

It is logical that the question arises as to whether NRP1 can be used as a therapeutic target, but it is too early to think about drug candidates at this stage of basic research. In the podcast, Drosten points out that NRP1 is useful for the basic state of the cell and the normal functioning of the body. An antibody or a small molecule is disturbed, this could also have consequences for the normal metabolism of the body. More intensive research is needed to demonstrate the exact role of neuropilin-1 in the Covid-19 transmission process.

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