SARS-Central CoV-2's Nervous System Targets and Routes

Mili Horton*

Editorial office, Journal of Neurology & Neurophysiology, Brussels, Belgium

Corresponding Author*

Mili Horton
Editorial office, Journal of Neurology & Neurophysiology, Brussels, Belgium
E-mail: Milihorton3@gmail.com

Received: 04-Nov-2022, Manuscript No. jnn-23-85311**; Editor assigned:** 09-Nov-2022, PreQC No jnn-23-85311 (PQ); **Reviewed:** 24-Nov-2022, QC No. jnn-23-85311 (Q); **Revised:** 29-Nov-2022, Manuscript No. jnn-23-85311 (R); **Published:** 30-Nov-2022,doi: 10.35248/2471-268X.22.13.11.604

Abstract

Neurological symptoms and indications show the presence of targets other than the major lung consequences of the Coronavirus Disease 2019 (COVID-19) pandemic. The SARS-CoV-2 coronavirus, which is the cause of COVID-19, demonstrates neurotropism for both the central and peripheral nervous systems. The virus can access the central nervous system by a variety of infective mechanisms and pathways, some of which skip the blood-brain barrier and others that compromise its integrity. There can be no doubt that the membrane-bound metalloprotease ACE2 serves as the SARS-CoV-2 host-cell receptor, according to a number of studies. The cellular location of the ACE2 enzyme in many tissues, including the central nervous system, has been examined using histochemical investigations and more recently, transcriptomics of mRNA.

Keywords: COVID-19 • Neurotropic virus • Angiotensin converting enzyme

Introduction

The Coronaviridae family of enclosed viruses contains the biggest single-stranded positive-sense RNA fragments ever discovered in an RNA virus, measuring between 26 and 32 kilobases. Many different species of birds and mammals are affected by Coronaviruses (CoVs). Most commonly, the first group of Human CoVs (HCoV-OC43, HCoV-293, HCoV-NL63, and HKU1-CoV) produces minor, self-limiting respiratory illnesses. The Middle East Respiratory Syndrome (MERS-CoV) and Severe Acute Respiratory Syndrome (SARS-CoV) viruses are part of the second group of HCoVs that are more dangerous and were the causes of the two outbreaks that occurred earlier this century. The severe acute respiratory syndrome coronavirus 2 virus, which was just identified as the seventh human CoV, is the source of the continuing Coronavirus Disease 2019 (COVID-19) outbreak (SARS-CoV-2).

Other viruses, like the betaCoV of the mouse hepatitis virus (MHV), rely on the S protein's N-term domain to attach to the cell adhesion molecule 1 that is related to carcinoembryonic antigen (CEACAM1). The host-cell receptor molecule for human H229E-CoV, transmissible gastroenteritis virus, porcine epidemic diarrhoea virus and feline infectious peritonitis virus is Aminopeptidase N (APN, CD13), a zinc metalloprotease. The unciliated bronchial epithelial cells' apical surface contains the enzyme Dipeptidyl-Peptidase 4 (DPP4), also known as a cluster of differentiation 26 (CD26), which serves as the MERS-CoV receptor. Using a distinct area of its spike protein S, MERS-CoV can also infect human pulmonary epithelial cells through interactions with sialic acid residues present in the host cell-surface glycoproteins.

Nasal mucosa viral infections in human

Nearly two decades ago, the two most common chronic medical conditions—rhinitis and rhinosinusitis, which can occasionally coexist with polyposis—were already affecting about 32 million people in the USA alone, with irreversible dysfunction most frequently seen in the elderly. The most frequent nontraumatic cause of olfactory dysfunction is acute viral rhinitis, but head injuries that cause tearing or severing of olfactory neuron axons at the cribriform plate are typically linked with traumatic causes.

The CNS has ACE2

The anti-inflammatory and hypotensive component of the renin-angiotensin-aldosterone system is mostly dependent on ACE2 (RAAS). In the CNS, an endogenous RAAS is active two branches make up this system, which is crucial for brain function: the vasoconstrictor and pro-inflammatory renin-angiotensin ACE branch and the vasodilator and anti-inflammatory ACE2-angiotensin Mas receptor arm. The vasodilator peptides, Ang and Ang, are produced by ACE2 by enzymatic modification of the vasoconstrictor peptides angiotensin II and angiotensin I. The most prevalent type of Angiotensin (Ang) is found in several parts of the brain, including the amygdala and hypothalamus. Speculative replacement SARS-CoV-2 can spread via additional channels besides those that start in the nasal mucosa.

The symptomatology of certain of neurological manifestations and/or consequences of COVID-19 can be connected with the broad distribution of the host-cell receptor for SARS-CoV-2 in the CNS. There are relatively few autopsy data available. SARS-CoV-2 RNA was found in the respiratory and cardiovascular regulatory centres of the medulla oblongata in a recent analysis of COVID-19 autopsy. Although some cases of severe encephalitis and encephalopathies as neurological consequences have been documented, further information is needed to fully substantiate the direct attack on the CNS or its participation as a cause of death [1-3].

Alternative hypothetical routes that SARS-COV-2 could take in order to enter the brain

- In 45% of COVID-19 necropsies, the initial hematogenous pathway, and evidence of gastrointestinal illness is seen. The effects of hyperimmune response syndrome on the endothelial cell bed (cytokine release syndrome) and/or adverse pre-existing endothelial conditions, as seen in several comorbidities in COVID-19 patients, may lay the groundwork for capillary dysregulation supporting SARS-CoV-2 infection of the CNS after defeating a weak BBB.
- Once SARS-CoV-2 has entered the general circulation, it may use a second hematogenous route, or the so-called "Trojan horse" mechanism, which is used by a number of microorganisms to infect the brain parenchyma. This route involves the extravasation of inflammatory phagocytic cells (leukocytes, primarily monocytes and lymphocytes) into the meninges and cerebrospinal fluid [4].
- A third possible neural pathway is that SARS-CoV-2 virions, after getting through the intestinal epithelial wall, could then directly infect the sympathetic neuron of the dorsal root ganglia as well as submucosal or myenteric plexus neuronal cells. After centripetally entering the CNS through the spinal cord, the viruses would be able to spread either synaptically across neurons or by crossing the blood-cerebrospinal fluid barrier and the choroid plexus, once more using the Trojan horse technique to get over the BBB [5].

Conclusion

The location and quantity of the ACE2 molecule in various cells control the viral load and possibly also the tropism of the virus on each target surface. evaluated the "cellular mapping" of the receptor molecule in the different

cell phenotypes of the nasal and intestinal mucosae using RNA-Seq analysis. The sustentacular cell, a non-neuronal epithelial cell, may be the most likely site of peripheral lesion associated with the dysosmias in COVID-19, although there is still no universal agreement on this. This is because of the topography and abundance of ACE2 and SARSCoV-2 coreceptor protein, TMPRSS2, as well as experiments in animal models. It travels to the CNS and other targets via the apical plasmalemma of the intestinal epithelial cell, the enterocyte, which has a much larger receptive zone. After attaching to ACE2 and fusing to the apical membrane of the enterocyte with the aid of TMPRSS2 and TMRSS4, the intestinal mucosa is suggested to be a favoured entrance point, primary viral reservoir, and preferable starting point for neurotropic routes that could be employed by SARS-CoV-2. Examples of the relationship between some clinical signs of COVID-19 and these proposed pathways are given.

References

1. Olender, et al. "The human olfactory transcriptome." BMC genom. 17.1 (2016): 1-18.
2. Åkerlund, A., et al. "Olfactory threshold and nasal mucosal changes in experimentally induced common cold." *Acta oto-laryngol.* 115.1 (1995): 88-92.
3. Yamagishi, M., and Y. Nakano. "A re-evaluation of the classification of olfactory epithelia in patients with olfactory disorders." *Eur. arch. Oto-rhino-laryngol.* 249.7 (1992): 393-399.
4. Suzuki, et al. "Identification of viruses in patients with postviral olfactory dysfunction." *Laryngoscope* 117.2 (2007): 272-277.
5. Van Riel, D., et al."The olfactory nerve: a shortcut for influenza and other viral diseases into the central nervous system." *J. pathol.* 235.2 (2015): 277-287.

Cite this article: Horton, M. SARS-Central CoV-2's Nervous System Targets and Routes. J Neuro Neurophysiol. 2022, 13 (11), 001-002