

Angiotensin-converting enzyme 2 receptor

Entry of [SARS-CoV-2](#) into human cells is dependent on the SARS-CoV receptor, angiotensin converting enzyme 2 ([ACE2](#)) receptor, and cathepsin. [Cathepsin](#) degrades the [spike protein \(S protein\)](#), which results in the entry of viral nucleic acid into the human host cell.

This [receptor](#) is also found in the [CNS](#) and plays a crucial role in autoregulating [cerebral perfusion pressure](#) ^{1) 2)}.

The triad of neuroinvasion of SARS-CoV-2, induction of hypercoagulable state, and the inhibition of [ACE2](#) blocking the formation of [Angiotensin](#) serve as the pathophysiology for neurovascular insults.

[SARS-CoV-2](#), which causes the [Coronavirus Disease 2019 \(COVID-19\)](#) pandemic, has a brain [neurotropism](#) through binding to the [Angiotensin-converting enzyme 2 receptor](#) expressed by [neurons](#) and [glial cells](#), including [astrocytes](#) and [microglia](#). Systemic infection which accompanies severe cases of COVID-19 also triggers substantial increase in circulating levels of [chemokines](#) and [interleukins](#) that compromise the [blood-brain barrier](#), enter the [brain parenchyma](#) and affect its defensive systems, [astrocytes](#) and [microglia](#). Brain areas devoid of a [blood-brain barrier](#) such as the circumventricular organs are particularly vulnerable to circulating inflammatory mediators. The performance of astrocytes and [microglia](#), as well as of immune cells required for brain health, is considered critical in defining the neurological damage and neurological outcome of COVID-19. In a review, they discussed the [neurotropism](#) of SARS-CoV-2, the implication of [neuroinflammation](#), adaptive and innate immunity, autoimmunity, as well as astrocytic and microglial immune and homeostatic functions in the neurological and psychiatric aspects of COVID-19. The consequences of SARS-CoV-2 infection during ageing, in the presence of systemic comorbidities, and for the exposed pregnant mother and foetus are also covered ³⁾

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