Protein mutation affects spread and virulence of respiratory virus

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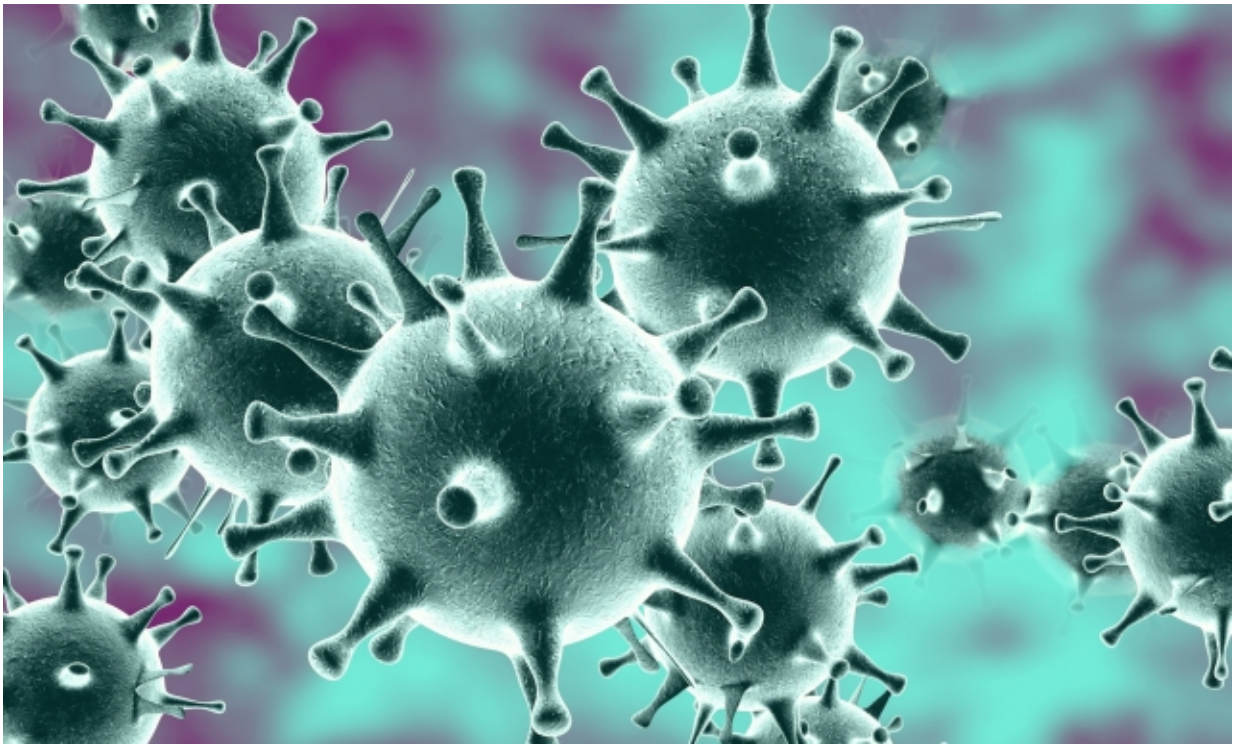

Quebec researchers have discovered that a mutation in a coronavirus protein slows the spread of the virus in the central nervous system and reduces its neurovirulence. It is the first time that this phenomenon has been observed in the coronavirus family, which is responsible for one-third of common colds and is also suspected of being associated with the

development or aggravation of neurological diseases such as multiple sclerosis, Alzheimer's disease, and encephalitis. The discovery, which has just been published in the prestigious journal *PLoS Pathogens*, was achieved in the Laboratory of Neuroimmunovirology at INRS-Institut Armand-Frappier.

In analyzing more than 60 human respiratory tract samples from patients infected by the human coronavirus, researchers discovered an important mutation in the S protein that modifies the virus capacity to infect nerve cells. The mutation is associated with the degree of viral virulence.

"We noticed that the protein mutation did not affect the virus's ability to infect the central nervous system, but that the mutated virus was less pathogenic and neurovirulent, probably as a result of changes in the way it spread from neuron to neuron due to the action of cellular proteins known as proprotein convertases, which alter the structure of the viral protein," explained lead researcher Professor Pierre Talbot. "Under these conditions, the coronavirus could more easily cause a persistent central nervous system infection. In virology, this phenomenon is known to trigger certain slow-developing neurological conditions or aggravate neurological diseases"

These results make it possible to better understand how persistent coronavirus infections take hold and may help prevent the development of associated neurological diseases in humans.

More information: Alain Le Coupanec et al. Cleavage of a Neuroinvasive Human Respiratory Virus Spike Glycoprotein by Proprotein Convertases Modulates Neurovirulence and Virus Spread within the Central Nervous System, *PLOS Pathogens* (2015). DOI: 10.1371/journal.ppat.1005261

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