



New Study Admits That COVID-19 Can Infect Brain Cells

Dr. Russell Blaylock, MD wrote in May that “By wearing a mask, the exhaled viruses will not be able to escape and will concentrate in the nasal passages, enter the olfactory nerves and travel into the brain.” That’s why I will never wear a mask! □ TN Editor

The coronavirus that causes COVID-19 can sometimes hijack brain cells, using the cells’ internal machinery to copy itself, according to a new study.

The research, posted Sept. 8 to the preprint database [bioRxiv](https://www.biorxiv.org/), has not yet been published in a peer-reviewed journal, but it provides evidence that SARS-CoV-2 can directly infect brain cells called neurons. Although the coronavirus has been linked to various forms of brain damage, from deadly inflammation to brain diseases known as encephalopathies, all of which can cause confusion, brain fog and delirium, there was little evidence of the virus itself invading brain tissue until now.

“We are actively looking at more patient tissues to be able to find how

frequently such brain infections occur ... and what symptoms correlate with infection of which areas of the brain,” senior author Akiko Iwasaki, an immunologist at Yale University, told Live Science in an email. In addition, scientists must still figure out how the virus enters the brain in the first place, and whether it can be kept out of the brain, the authors noted in their report.

Human, mouse and mini-brains

To see whether SARS-CoV-2 could break into brain cells, the study authors examined autopsied brain tissue from three patients who died of COVID-19. They also conducted experiments in mice infected with COVID-19 and in organoids — groups of cells grown in a lab dish to mimic the 3D structure of brain tissue.

“This study is the first to do an extensive analysis of SARS-CoV-2 [brain] infection using three models,” said Dr. Maria Nagel, a professor of neurology and ophthalmology at the University of Colorado School of Medicine, who was not involved in the study. Previously, there were only “rare case reports” of SARS-CoV-2 RNA and viral particles found in post-mortem tissue from patients, Nagel, who specializes in neurovirology, told Live Science in an email.

In the organoids, the team found that the virus could enter neurons through the ACE2 receptor, a protein on the cell surface that the virus uses to enter the cell and trigger infection. They then used an electron microscope, which uses beams of charged particles to illuminate the tissue, to peer inside infected cells. They could see coronavirus particles “budding” within the cell, demonstrating that the virus had commandeered the neurons’ internal machinery to build new copies of itself.

While setting up shop in infected cells, the virus also caused metabolic changes in nearby neurons, which were not infected. These nearby cells died off in large numbers, suggesting that the infected cells might steal oxygen from their neighbors in order to keep producing new virus, the authors noted.

“We do not know if similar events are taking place in infected people,” though there is some evidence they might be, Iwasaki noted. In the autopsied tissue, the team found SARS-CoV-2 had infected some neurons in the wrinkled cerebral cortex. Near these infected cells, they found evidence of “small strokes” having taken place, hinting that the virus might steal oxygen from nearby cells in the brain just as it did in the organoids, Iwasaki said.

Notably, the infected brain tissue was not flooded with immune cells, as might be expected. When the Zika virus or rabies virus invades the brain, a large number of immune cells usually follow, the authors noted. So it’s possible that when SARS-CoV-2 manages to infiltrate the brain, it may somehow escape the body’s typical defense against such invasions. It’s not yet known how this unusual immune response might affect the course of the infection, but it may make the virus more difficult to clear from the brain. And though few immune cells flock to the site of infection, dying neurons nearby can trigger a chain-reaction in the nervous system that still leads to harmful inflammation, the authors noted.

Finally, in the mouse experiments, the authors genetically modified one group of mice to express human ACE2 receptors in their brains, while another group of mice only bore the receptor in their lungs. The first set of mice rapidly began losing weight and died within six days, while the second set did not lose weight and survived. In addition, in the mice with brain infection, the arrangement of blood vessels in the brain changed dramatically, presumably to redirect nutrient-rich blood to “metabolically active hot spots” where the virus had taken over, the authors wrote.

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