Tulane study reveals key differences in long-term impacts of COVID-19 and flu

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A Tulane University study found that COVID-19 caused brain inflammation and disruption of serotonin and dopamine regulation pathways in mice, potentially explaining why some feel brain fog after a bout with the virus. (Photo by iStock)

Even a mild case of COVID-19 or the flu can impact the body long after the fever and cough fade, according to new Tulane University research that may help explain why some people struggle to feel fully recovered weeks or months later.

Tulane researchers found that while both viruses can leave lasting lung damage, only SARS-CoV-2 infection caused persistent brain inflammation and small blood vessel injury, even after the virus was no longer detectable. The findings, published in Frontiers in Immunology, help explain why long COVID often includes neurological symptoms such as brain fog, fatigue and mood changes, while influenza is more commonly associated with respiratory complications.

“Influenza and COVID-19 affect large populations worldwide and carry a significant public health toll, yet the mechanisms behind their long-term effects remain poorly understood,” said Dr. Xuebin Qin, lead author and professor of microbiology and immunology at the Tulane National Biomedical Research Center.

To separate effects common to severe respiratory infections from those unique to COVID-19, researchers used a mouse model to examine lung and brain tissue after infection had cleared.

In the lungs, both viruses left behind a similar picture: immune cells that failed to fully stand down and increased buildup of collagen, a protein associated with scarring. Those changes can stiffen lung tissue and make breathing feel more labored — a possible biological explanation for why some people report lingering shortness of breath after respiratory infections.

But when the researchers looked more closely, they found a key difference. After the flu, the lungs appeared to switch into repair mode, sending specialized cells into damaged areas to help rebuild the lining of the airways. That repair response was largely missing after COVID-19 infection, suggesting the virus may interfere with the lung’s natural healing process.

The most striking differences appeared in the brain.

Although neither virus was found in brain tissue, mice that had COVID-19 showed signs of persistent brain inflammation weeks later, along with tiny areas of bleeding. Gene expression analysis revealed ongoing inflammatory signaling and disruption of pathways involved in serotonin and dopamine regulation, systems closely tied to mood, cognition and energy levels. These persistent changes were largely absent in influenza-infected animals.

“In both infections, we observed lasting lung injury,” Qin said. “But long-term effects in the brain were unique to SARS-CoV-2. That distinction is critical to understanding long COVID.”

This study was supported by an American Heart Association award Qin received as part of a national effort to understand the long-term cardiovascular and cerebrovascular effects of COVID-19. The findings shed new light on how vascular and immune changes may contribute to persistent neurological symptoms.

By defining these biological changes, the research offers a clearer foundation for monitoring patients and developing treatments aimed at preventing lasting damage. As lingering symptoms continue to complicate recovery for some, understanding what is driving them is essential to reducing long-term health consequences.

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