

Yale Study Links Some Long COVID Patients to Autoimmune Responses

Antibodies from some long COVID patients attacked brain and nerve tissues, a finding that could point toward treatments.

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Since the COVID-19 pandemic first swept the world in 2020, the chronic condition known as long COVID has baffled the medical community. In the years since, scientists have tried to determine why a subset of patients infected with the virus develop lingering — and sometimes disabling — symptoms that can last long after the initial SARS-CoV-2 infection.

[A new study](#) co-led by Yale's [Akiko Iwasaki](#) yields intriguing new insights.

[Writing in the journal Cell](#), the team of researchers report strong evidence that, in at least a subset of people with long COVID, the body's immune system launches an autoimmune attack against the patient's own cells, causing a cascade of symptoms such as fatigue, brain fog, weakness, and other hallmarks of the condition. Autoimmune disorders occur when the body's immune system attacks its own tissues.

The findings suggest that long COVID may overlap with autoimmune diseases, even though it doesn't perfectly resemble any known autoimmune condition, said Iwasaki, Sterling Professor of Immunobiology at Yale School of Medicine (YSM) and co-senior author of the study.

"This is a significant finding, but that doesn't mean there aren't other causes," said Iwasaki, who has been a leading investigator on COVID immune response. "Our study does not explain the entire long COVID scenario. This is one possible cause of long COVID, but it will likely have other trigger causes as well."

Still, if eventually validated, the study could point toward future long COVID treatments, including therapies already used for other autoimmune diseases, although the researchers stress that much more investigation is needed.

Iwasaki is also a professor of dermatology and of molecular, cellular, and developmental biology in Yale's Faculty of Arts and Sciences, a professor of epidemiology at Yale School of Public Health, and an investigator of the Howard Hughes Medical Institute.

Since long COVID first emerged six years ago, researchers in Iwasaki's lab have been chipping

away at the mysterious condition. Its causes have been difficult to pin down because symptoms vary widely from patient to patient. But the new findings provide some of the strongest evidence so far that autoimmunity may be one important factor.

For the study, the Yale team and collaborators — including a Mount Sinai Health System team led by study co-senior author David Putrino, a professor of rehabilitation and human performance and the Nash Family Director of the Cohen Center for Recovery from Complex Chronic Illness — focused specifically on autoantibodies, immune proteins that are known to mistakenly target the body's own tissues instead of viruses or bacteria.

The researchers discovered that many long COVID patients had autoantibodies aimed at parts of the brain and nervous system.

These autoantibodies frequently targeted tissues involved in pain signaling, memory, balance, sensory processing, and autonomic nervous system control, they found, which could help explain long COVID symptoms such as brain fog, dizziness, headaches, fatigue, burning pain, and numbness.

After analyzing blood samples from people with long COVID, from healthy volunteers, and from people who had recovered from COVID but had not developed lasting symptoms, the researchers purified antibodies from patients' blood and exposed them to human and mouse tissues. They found that antibodies from long COVID patients reacted more strongly with certain brain regions and nerve tissues than antibodies from control groups.

The researchers then screened these blood samples against more than 21,000 human proteins to identify what the antibodies were targeting. Many of the targets, they found, were linked to neurons, nerve communication, inflammation, and hormone signaling.

Finally, they transferred antibodies from long COVID patients into healthy mice. The researchers conducted various behavioral studies working with a team led by study co-senior author Tamas Horvath, the Jean and David W. Wallace Professor of Comparative Medicine, professor of neuroscience and of obstetrics, gynecology, and reproductive sciences at YSM. They observed that the mice developed increased pain sensitivity, fatigue, impaired balance, and damage to small nerve fibers.

Working with Marc Schneeberger Pane, assistant professor in cellular and molecular physiology in Yale's Faculty of Arts and Sciences, the team found that mice also showed abnormal neuronal activation in brain regions involved in pain, fatigue, memory, and emotional regulation.

"What was most fascinating about this work is that we were able to find antibodies that, when transferred to mice, caused the same type of symptoms that are reported by long COVID patients," said study lead author Keyla Santos Guedes de Sá, a postdoctoral associate in immunobiology in Iwasaki's lab.

Researchers will next examine how the damage is being done.

“Now that we were able to identify a subgroup of patients whose condition might be driven by autoantibodies, we want to investigate the neurological and immunological mechanisms by which these autoantibodies are causing disease,” said Sá.

The rise of long COVID itself wasn’t a surprise, Iwasaki added.

“When you dig into the literature, you see that every major pandemic is accompanied by a long-version, chronic illness that follows,” she said. “It happens when a human population is exposed to a new pathogen — or sometimes not even a new pathogen but an existing one like EBV [Epstein-Barr virus.] Many viral pathogens are capable of triggering these chronic diseases after infection.”

For researchers, discovering effective treatments is the end goal.

“We’re all — Keyla and I and everybody in our team — driven by the desire to help people with long COVID,” Iwasaki said. “Right now, there is no approved treatment for these people, and they really need help.”

Other Yale researchers involved in the study included scientists from the Department of Immunobiology in YSM, the Center for Infection and Immunity, the Department of Comparative Medicine, the Department of Cellular and Molecular Physiology, and the Department of Pathology. Other partners included researchers from the Yale Cancer Biology Institute on West Campus and from the Department of Biostatistics at the Yale School of Public Health.

In addition to scientists from the Icahn School of Medicine at Mount Sinai, other partners included researchers from the Howard Hughes Medical Institute; Serom Yx Systems; CellTrend GmbH in Germany; and the Institute of Medical Immunology, Charité - Universitätsmedizin Berlin, Corporate Member of Freie Universität Berlin and Humboldt Universität zu Berlin and Berlin Institute of Health.

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