

GLP-1 Agonists May Help People With Diabetes and Long COVID

Mice treated with GLP-1 drugs showed reduced pulmonary fibrosis compared to those not treated with GLP-1 agonists, suggesting that the drug could lessen the risk of fibrous buildup.

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Washington, D.C.—GLP-1 agonists have become popular treatments for weight loss and type 2 diabetes, and newer studies suggest other health benefits as well. Findings from experiments on mice, published this week in the [Journal of Virology](#), suggest that the drugs may offer benefits to another patient population: people with type 2 diabetes who suffer from pulmonary fibrosis months or years after infection with SARS-CoV-2, the virus that causes COVID-19.

“People with diabetes have way more severe infections and infection-related severe disease after COVID-19 than other people,” said virologist and study leader Runhong Zhou, PhD, at the University of Hong Kong, who studies interactions between viruses and the immune system. Studies have estimated that people with diabetes face a risk of severe long COVID symptoms that is four times higher than people who don’t have diabetes, he said, but the mechanisms aren’t well understood. “We wanted to know why these people have long-term symptoms.”

Pulmonary fibrosis is a lung disease characterized by widespread scarring, primarily caused by immune cells called macrophages. It is among the most severe symptoms of long COVID, a condition describing myriad, persistent health problems that can linger after COVID-19 infection. The thickened lung tissue inhibits the diffusion of oxygen into the bloodstream and can cause severe coughing and other symptoms.

During this study, Zhou was a researcher in the lab of virologist Zhiwei Chen, PhD, also at the University of Hong Kong, who has been studying immune responses and immunopathogenesis of coronaviruses in animals since a SARS outbreak in China that began in November 2002.

To better understand how type 2 diabetes contributes to pulmonary symptoms in long COVID, Zhou and his collaborators first compared blood samples, used in a previous study on COVID-19, from people with and without diabetes at three distinct time points after admission to a hospital for treatment. At the third time point, months after hospitalization, the researchers found differing levels of monocytes, which are white blood cells that can develop into macrophages. In people with diabetes, they also identified increased activity in genetic pathways involving monocytes that

are linked to the onset of fibrosis.

Then, they used experiments on mice to confirm the connections among diabetes, SARS-CoV-2 infection and the onset of pulmonary fibrosis. Those experiments showed, for example, that numerous fibrosis-related genes—which under normal conditions are responsible for fibrous proteins the body needs—were overly active in the mice, as they had been in infected people. The next step, said Zhou, was to find a way to interfere with those pathways.

“We wanted to figure out what kind of treatment may prevent this in the mouse,” Zhou said. The GLP-1 receptor is highly expressed in lung cells, he said, and during the study, he saw other laboratory experiments reporting that GLP-1 agonists showed some anti-inflammatory effect against fibrosis related to pneumonia. However, he hadn’t seen it tested in animal models after severe COVID-19 infection.

They found that treating the mice with GLP-1 agonists normalized the fibrosis-related genes, “reprogramming” the macrophages that caused the scar tissue buildup. Treated mice showed reduced pulmonary fibrosis compared to those not treated with GLP-1 agonists, suggesting that the drug could lessen the risk of fibrous buildup.

Zhou cautioned that the new findings are preliminary. They show proof of concept and only involved a small number of mice, but the findings gave him optimism that the treatment is worth investigating.

Though the global emergency spurred by COVID-19 has lessened, long-term health problems continue to plague many people.

“Long COVID is a continuous issue, and especially a lot of elderly people are still suffering from this,” Chen said, who noted that his lab developed a suite of antibody drugs that show promise against the condition. “So, to find a solution to help these patients is an important issue.”

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