# Tulane researchers receive American Heart Association grant to study COVID's vascular effects

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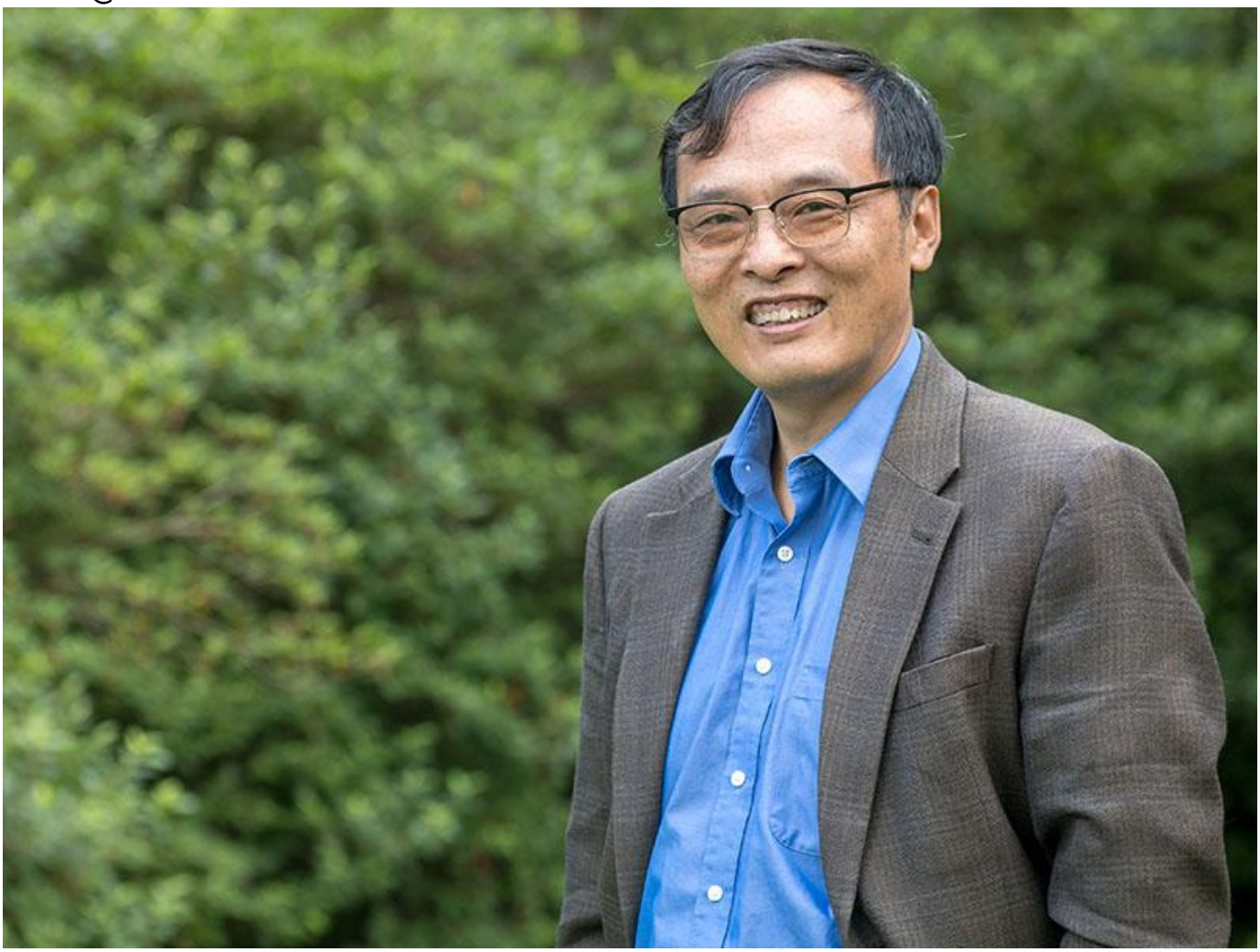

Dr. Xuebin Qin, professor of microbiology and immunology at the Tulane National Primate Research Center.

The American Heart Association has awarded Tulane University researchers $940,000 to study how COVID-19 spurs vascular inflammation that may increase risks for blood clots and lingering symptoms of long COVID.

The grant is one of 11 recently awarded by the American Heart Association as part of a $10 million effort to study the cardiovascular and cerebrovascular impacts of COVID-19 as the pandemic enters its third year.

Tulane researchers will investigate the role that endothelial cells play in the development of severe and long COVID-19. Endothelial cells line blood vessel walls throughout the body and can malfunction following SARS-CoV-2 infection. This dysfunction can cause over-coagulation and blood clotting in major organs such as the heart and lungs, as seen in the most severe COVID cases. It may also contribute to complications associated with long COVID.

Dr. Xuebin Qin, professor of microbiology and immunology at the Tulane National Primate Research Center, will lead a cross-disciplinary team with the Tulane University School of Medicine to explore how the dysfunction of endothelial cells contributes to severe and long COVID outcomes.

The team developed transgenic mouse models genetically engineered to simulate COVID in the way that humans experience the most severe outcomes of the disease, including the development of ARDS, or Acute Respiratory Distress Syndrome.

In the human population, those who experienced ARDS also had endothelial cell injury, causing a weakening of the “tight junctions” that normally serve to connect endothelial cells. If these junctions weaken and leak, blood can escape and pool in the tissues of major organs, causing abnormal clotting and tissue damage.

It is the dysregulation of these tight junctions, they propose, that contributes to severe and long COVID outcomes, particularly those related to the vasculature of the heart and brain.

“The health outcomes associated with COVID extend far beyond the respiratory system,” Qin said. “By understanding how SARS-CoV-2 infection triggers endothelial cell dysfunction, we hope to open the door to new therapies that prevent the cascade of effects that we see in the most severe and lasting COVID cases.”

Qin is the principal investigator; co-investigators include Dr. Jay Kolls, John W Deming Endowed Chair in Internal Medicine; Dr. Patrice Delafontaine, professor of medicine, pharmacology and physiology; Yusuke Higashi, PhD, associate professor of medicine; Nicholas Maness, PhD, associate professor of microbiology and immunology; and Stephen Braun, PhD, assistant professor of pharmacology.