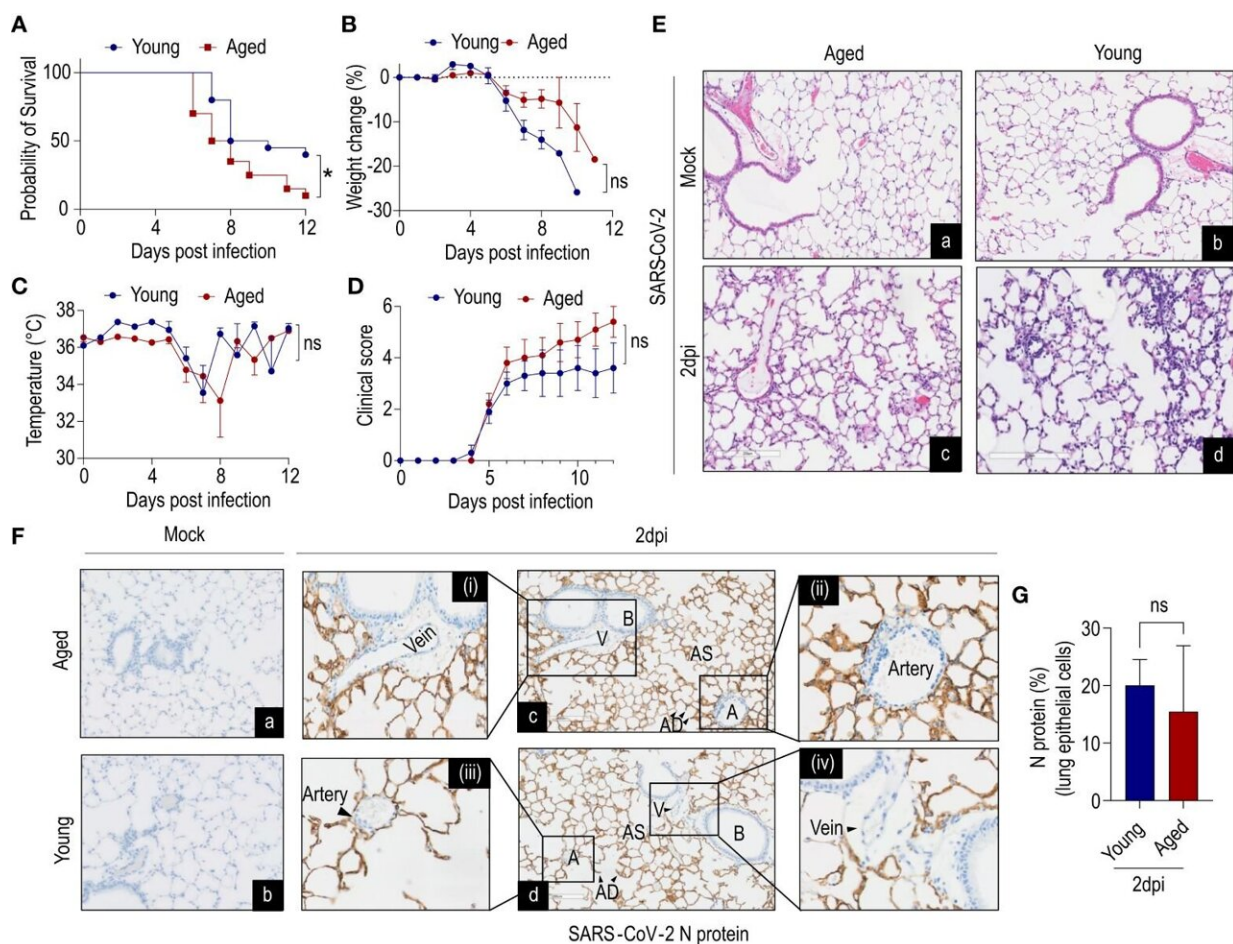


New study helps explain how elderly individuals react differently to COVID-19 than young people

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Age-dependent clinical decline of SARS-CoV-2-infected humanized ACE2 mice. K18-hACE2 mice young (12–15 weeks) and aged (85–112 weeks) were inoculated intranasally with 1×10^4 plaque-forming units (PFU) or received saline (mock). Credit: *Frontiers in Immunology* (2024). DOI:

10.3389/fimmu.2024.1397990

The COVID-19 pandemic resulted in over 700 million infections and 7 million deaths worldwide. While age is recognized as a risk factor for severe COVID-19, the reasons for this are not yet fully understood.

A new study by researchers at Boston University Chobanian & Avedisian School of Medicine suggests that the immune response of lung endothelial cells, which line the [blood vessels](#), is too low during the early stage of COVID-19 infection as demonstrated in a preclinical model. Additionally, the researchers analyzed all genes expressed in purified endothelial cells, which had never been done before.

"The susceptibility to SARS-CoV-2 infection increases proportionally with age, placing older individuals at a significantly higher risk of developing severe COVID-19. Therefore, gaining insight into age-dependent pathological changes during SARS-CoV-2 infection is imperative for effectively safeguarding vulnerable populations," says corresponding author Markus Bosmann, MD, associate professor of medicine, pathology & laboratory medicine at the school.

The researchers used four groups of endothelial cell conditions with susceptibility to SARS-CoV-2. The first two groups, consisting of young and old models, remained uninfected as controls. The other two groups, also young and old, were infected with SARS-CoV-2.

Endothelial cells from all sets of conditions were isolated after two days, and their transcriptomes (their expressed [genes](#)) were analyzed and classified as biological programs of the host response. The clinical severity of infection was monitored and found to be more severe with advanced age.

According to Bosmann, a suppressed immune landscape is a key driver of age-associated [endothelial dysfunction](#) during COVID-19. "While these findings currently do not have immediate implications for treating COVID-19, targeting these immune pathways in [endothelial cells](#) may have prognostic and therapeutic benefits although further studies, including dissecting these functional changes at a single-cell level, are needed," he adds.

These [findings](#) appear online in the journal *Frontiers in Immunology*.

More information: Aging is associated with an insufficient early inflammatory response of lung endothelial cells in SARS-CoV-2 infection, *Frontiers in Immunology* (2024). [DOI: 10.3389/fimmu.2024.1397990](#). [www.frontiersin.org/journals/immunology/2024/1397990/full](#)

Provided by Boston University School of Medicine

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