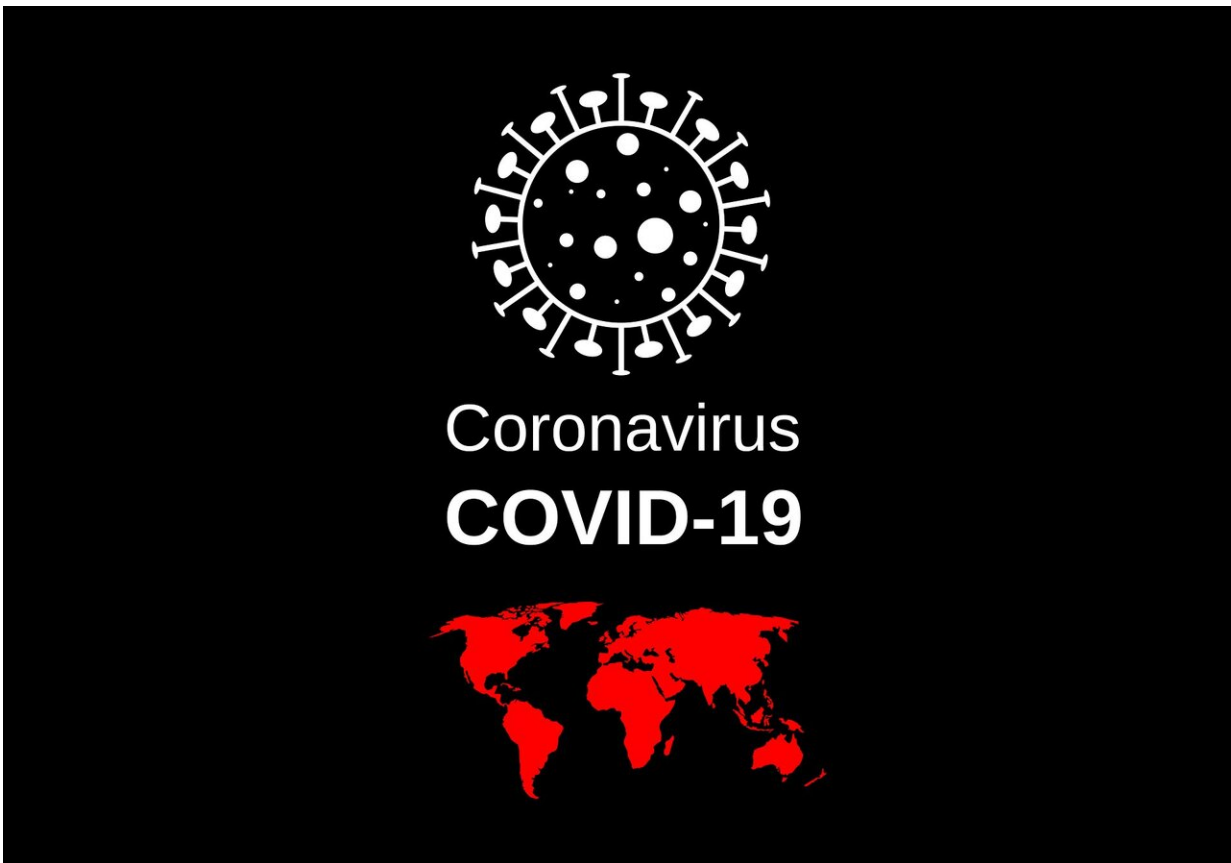


Study finds high blood pressure medications safe for patients with COVID-19 disease

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Despite concerns expressed by some experts, common high blood pressure drugs did not increase the risk of contracting COVID-19—or of developing severe disease—in a study of 12,594 patients.

Published online May 1 in the *New England Journal of Medicine*, the study was launched in response to a March 17 joint statement issued by the American Heart Association, the American College of Cardiology, and the Heart Failure Society of America. It urgently called for research to answer a question raised by past studies: do [high blood pressure](#) (antihypertensive) drugs worsen COVID-19 patient outcomes?

Led by researchers from NYU Grossman School of Medicine, the study found no links between treatment with four drug classes—angiotensin-converting enzyme (ACE) inhibitors, [angiotensin receptor blockers](#) (ARBs), beta blockers, or [calcium channel blockers](#)—and increased likelihood of a positive test for COVID-19.

Further, the study found no substantial increase in risk for more [severe illness](#) (intensive care, use of a ventilator, or death) with any of the treatments in patients with the pandemic virus.

"With nearly half of American adults having high blood pressure, and heart disease patients more vulnerable to COVID-19, understanding the relationship between these commonly used medications and COVID-19 was a critical public health concern," says lead investigator Harmony Reynolds, MD, associate director of the Cardiovascular Clinical Research Center at NYU Langone Health. "Our findings should reassure the [medical community](#) and patients about the continued use of these commonly prescribed medications, which prevent potentially severe heart events in their own right."

For the study, the researchers identified patients in the NYU Langone Health electronic health record with COVID-19 test results. For each identified patient with COVID-19 test results, the team discretely extracted medical history needed for the analysis, which compared treated and untreated patients.

"Before our study, there were no experimental or clinical data demonstrating the consequences of using these medications one way or the other in people at risk for COVID-19," says senior study author Judith Hochman, MD, the Harold Snyder Family Professor of Medicine and Senior Associate Dean for Clinical Sciences at NYU Langone Health. "In terms of next steps, our plan is to use similar approaches to investigate other medications and their relationship to COVID-19 illness."

Cause for Concern

The study revolves around drugs that act on the renin-angiotensin-aldosterone hormonal system, which influences blood pressure. Central to this system is the signaling protein angiotensin II, levels of which are controlled by angiotensin-converting enzyme (ACE), say the authors. Angiotensin II narrows blood vessels to increase blood pressure, and the study drugs counter that, either by blocking ACE-induced increases in angiotensin II, or the ability of ACE to interact with its receptor signaling partners on cells.

According to the researchers, one version of ACE, angiotensin converting enzyme 2 (ACE2), is present in the outer membrane of lung cells. SARS-CoV-2, the current pandemic virus, has been shown to connect to ACE2 on lung cells, a first step toward viral infection. This led to concern in the field that ACE inhibitors and ARBs might increase or worsen COVID-19 infection. Past studies in animal models had suggested that ACE inhibitors and ARBs increase ACE2 production in other organs, but how they related to ACE2 levels in the lungs was not known.

On the other hand, ACE inhibitors and ARBs had been shown elsewhere to reduce lung injury in certain viral pneumonias, creating speculation that they might be helpful. The new study was designed to address these

contradictions.

More information: Harmony R. Reynolds et al.
Renin–Angiotensin–Aldosterone System Inhibitors and Risk of
Covid-19, *New England Journal of Medicine* (2020). [DOI:
10.1056/NEJMoa2008975](https://doi.org/10.1056/NEJMoa2008975)

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