

Popular hypertension drugs don't increase risk of COVID-19 severity, fatality

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A new Northwestern Medicine study in mice found a widely used class of drugs to treat patients with hypertension, cardiovascular disease and diabetic kidney disease—many of whom are elderly—does not increase the risk of developing a severe and potentially fatal COVID-19 infection, as previously feared.

There have been concerns by the medical community worldwide that the drugs—ACE inhibitors and [angiotensin receptor blockers](#) (ARB)—might have caused an increase in ACE2, the main receptor for SARS-CoV-2, which could possibly increase the risk for this infection and its severity.

But the new findings revealed a decrease, not an increase, in ACE2 in mice kidney membranes and no change in lung membranes. The study supports the safety of these drugs in the face of the COVID-19 pandemic.

This study is the first to examine the effect of ACE2 and ARBs in the lungs, which are considered one of the main targets for SARS-CoV-2 entry into the body.

"This study supports the concept that there is no increased risk for COVID-19 infection by using ACE inhibitors and angiotensin receptor blockers," said Daniel Battle, the Earle, del Greco, Levin Professor of Medicine at Northwestern University Feinberg School of Medicine and a Northwestern Medicine nephrologist.

The paper was published recently in the *Journal of the American Society of Nephrology*.

ACE inhibitors and ARBs are a category of drugs called RAS blockers. These drugs, by different mechanisms, block the actions of a peptide that causes narrowing of blood vessels and fluid retention by the kidneys, which result in increased blood pressure. The drugs help blood vessels relax

and expand and decrease fluid retention, both of which lower blood pressure.

To examine this issue, Northwestern Medicine scientists measured ACE2 in isolated kidney and lung membranes of mice that were treated with either captopril, a widely used ACE inhibitor, or telmisartan, an ARB also widely prescribed.

Since the recognition that ACE2 is the main receptor for SARS-CoV-2, there have been multiple studies discussing the potential risk (or lack of) for susceptibility and worse clinical course of COVID-19 in patients treated with RAS blockers. Much of the speculation comes from previous animal studies where some RAS blockers were reported to upregulate ACE2 in the heart and kidney vasculature.

"My lab has long worked with ACE2, and this was a critical question that needed to be addressed," Battle said. First author Jan Wysocki said, "We had no bias one way or another, and the kidney findings showing lower ACE2 in treated animals were a bit unexpected."

More information: Jan Wysocki et al. Kidney and Lung ACE2 Expression after an ACE Inhibitor or an Ang II Receptor Blocker: Implications for COVID-19, *Journal of the American Society of Nephrology* (2020). [DOI: 10.1681/ASN.2020050667](#)

Provided by Northwestern University

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