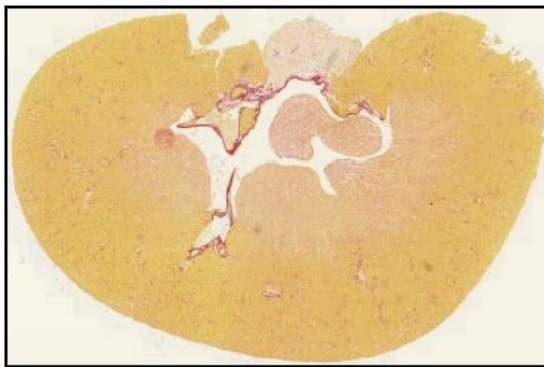


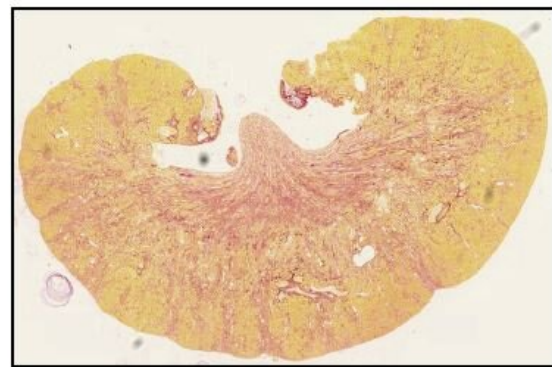
Drug discovery offers potential treatment for common kidney disease

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Contralateral kidney



IRI kidney (28 min) at 4 weeks



Scanned images of whole kidneys comparing a healthy contralateral mouse kidney with a kidney after ischemia-reperfusion injury (IRI), with red staining indicative of fibrosis. Credit: *Science Translational Medicine* (2022). DOI: 10.1126/scitranslmed.abf5074

A serious condition that can cause the kidneys to suddenly stop working could be treated with existing medicines, a new study shows.

In a study in mice, scientists found that medicines usually used to treat angina and high blood pressure prevented much of the long-term damage to the kidney and cardiovascular system caused by [acute kidney injury](#) (AKI).

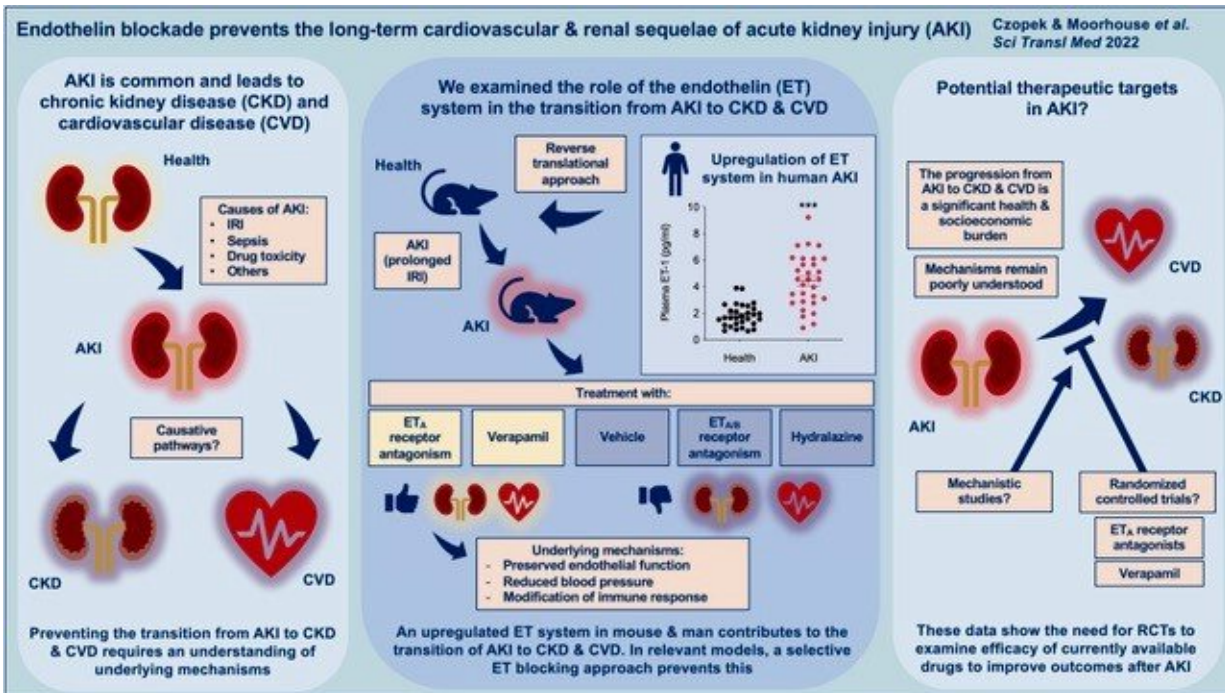
Experts hope the findings will pave the way for improved treatment of AKI—a common illness that occurs in approximately 20% of emergency hospital admissions in the U.K.

The condition is usually caused by other illnesses that reduce blood flow to the kidney, or due to toxicity arising from some medicines.

AKI must be treated quickly to prevent death. Even if the kidneys recover, AKI can cause long lasting damage to the kidneys and the cardiovascular system.

Of those who survive an episode of AKI, 30% are left with [chronic kidney disease](#) (CKD). The remaining 70% that recover full [kidney function](#) are at an almost 30-fold increased risk of developing CKD.

A team from the University of Edinburgh found that patients with AKI had increased blood levels of [endothelin](#)—a protein that activates inflammation and causes blood vessels to constrict. Endothelin levels remained high long after kidney function had recovered.



Endothelin blockade prevents the long-term cardiovascular & renal sequelae of acute kidney injury. Credit: *Science Translational Medicine* (2022). DOI: 10.1126/scitranslmed.abf5074

After finding the same increase in endothelin in mice with AKI, experts treated the animals with medicines that block the endothelin system. The medicines, normally used to treat angina and [high blood pressure](#), work by stopping the production of endothelin or by shutting off endothelin receptors in cells.

The mice were monitored over a four-week period after AKI. Those that were treated with the endothelin-blocking medicines had [lower blood pressure](#), less inflammation and reduced scarring in the kidney.

Their [blood vessels](#) were more relaxed and kidney function was also improved, compared with untreated mice.

The study is published in *Science Translational Medicine*.

Dr. Bean Dhaun, senior clinical lecturer and honorary consultant nephrologist at the University of Edinburgh's Center for Cardiovascular Science, said, "AKI is a harmful condition, particularly in [older people](#) and even with recovery it can have a long-term impact on a person's health. Our study shows that blocking the endothelin system prevents the long-term damage of AKI in mice. As these medicines are already available for use in humans, I hope that we can move quickly to seeing if the same beneficial effects are seen in our patients."

Professor James Leiper, associate medical director at the British Heart Foundation, said, "Impaired kidney function that results from acute kidney injury can also increase a person's chance of developing and dying from heart and circulatory diseases, so it's vital we find ways to reduce this risk.

"This promising research suggests that widely available medicines could help to tackle the impact of acute kidney injury before it can cause damage and further complications. While further studies will be needed to demonstrate whether this treatment is safe and effective for patients, this early research is an encouraging first step."

More information: Alicja Czopek et al, Endothelin blockade prevents the long-term cardiovascular and renal sequelae of acute kidney injury in mice, *Science Translational Medicine* (2022). [DOI: 10.1126/scitranslmed.abf5074](https://doi.org/10.1126/scitranslmed.abf5074).
www.science.org/doi/10.1126/scitranslmed.abf5074

Provided by University of Edinburgh

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