

# Protective molecule, ACE2, also proving its worth in diabetic patients

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ACE2, a molecule that has been shown to prevent damage in the heart, is now proving to be protective of the major organs that are often damaged in diabetic patients.

Gavin Oudit, a researcher with the Faculty of Medicine & Dentistry, and his colleagues at the University of Florida, found that lab models that lacked ACE2 had worse cardiovascular complications related to diabetes.

"We show that if you take ACE2 away, they [lab models of diabetes] do very poorly," said Oudit. "It worsened their [heart](#) function and their vascular function." "In patients, if you have high levels of ACE2 in your vascular reparative cells you do not get diabetic complications, even if your diabetic control is very poor."

The study was funded by the National Institutes of Health, Alberta Innovates – Health Solutions and the Canadian Institutes of Health Research.

Oudit's related studies have also looked at another major organ affected by diabetes – the kidneys. ACE2 proved protective in these organs, as well.

"Diabetes is the world's most common cause of blindness and renal failure, so this work is very important," he said.

The next step is to analyze human blood samples from Edmonton's patient population to see the effects of ACE2 in diabetics. The researchers will screen the blood of both Type 1 and Type 2 [diabetic patients](#) since they think it's relevant in both.

"We're hoping to show that patients that lack ACE2 are more susceptible to diabetic complications," said Oudit.

Human recombinant ACE2, which is a pure form of this enzyme, is currently moving into Phase 2 clinical trials as a treatment for lung disease. The hope is that its use could then be extended as a pharmaceutical to treat diabetic patients so they can avoid cardiovascular, renal or eye disease.

Oudit's work is published in the May 11th edition of the journal *Circulation Research* with an accompanying editorial.

Provided by University of Alberta Faculty of Medicine & Dentistry

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