Targeting diabetic kidney disease

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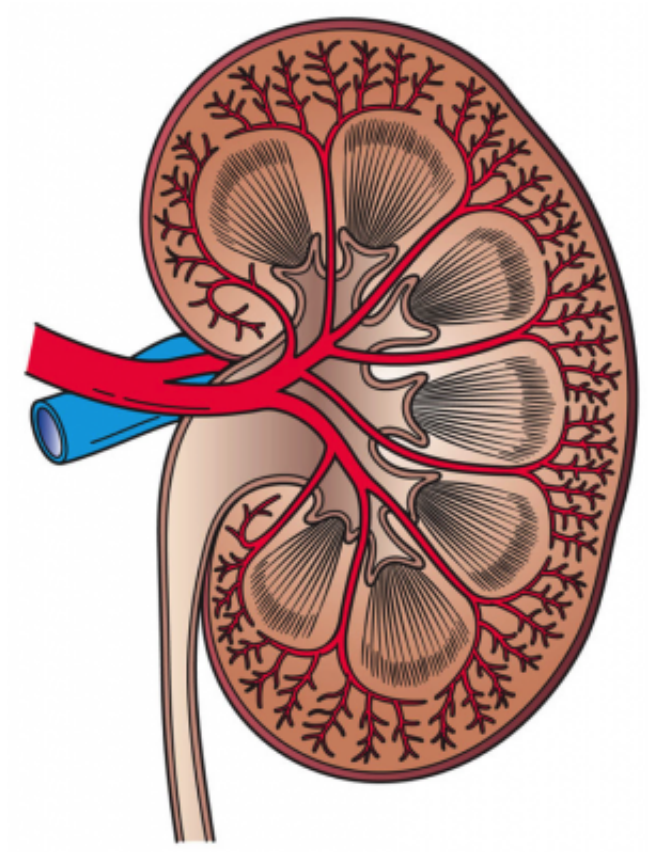

This image shows a cross section of a kidney. Credit: Holly Fischer/Wikipedia

Diabetic nephropathy (DN) is a kidney disease characterized by loss of kidney function in patients with diabetes.

Raymond Harris, MD, and colleagues previously showed that the epidermal growth factor receptor (EGFR) is activated in mouse models

of type 1 diabetes, and that drugs which inhibit EGFR prevent the development of DN.

EGFR becomes active when phosphate molecules attach at specific tyrosine sites in the protein. The phosphorylated tyrosines become the docking sites for a variety of molecules that regulate cell proliferation, maturation and cell death.

Now in a study published in the journal *Diabetes*, Harris, Ming-Zhi Zhang, MD, and colleagues show that blocking EGFR tyrosine kinase activity with the drug erlotinib not only slowed the progression of DN but also improved weight gain and reduced insulin resistance in mice. Their work suggests that pathways activated by EGFR may be attractive targets to treat DN.

For his discovery of EGF, Vanderbilt's Stanley Cohen, Ph.D., shared the Nobel Prize in medicine in 1986.

More information: Zhilian Li et al. Inhibition of Epidermal Growth Factor Receptor Activation Is Associated With Improved Diabetic Nephropathy and Insulin Resistance in Type 2 Diabetes, *Diabetes* (2018). DOI: 10.2337/db17-1513

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