

## **Decoding Diabetic Kidney Disease**

A research team at the [Icahn School of Medicine at Mount Sinai](#) has revealed biological pathways involved in diabetic kidney disease. They hope that with these new pathways, early diagnostic tests and targeted treatments can be designed. According to the National Kidney Foundation, about 30 percent of patients with type 1 diabetes and 10 to 40 percent of those with type 2 diabetes will eventually have end-stage renal disease (ESRD). The study focused on the kidney's glomerulus, which act as the key unit for blood filtration.

Researchers studied three different cell types, using two sets of mice. One group naturally developed diabetic kidney disease and the other was naturally resistant. In the mice prone to kidney disease, endothelial cells were affected. [Endothelial cells](#) form a single cell layer that lines all blood vessels and regulates exchanges between the bloodstream and surrounding tissues. In these cells the mitochondria, or energy units, of the cells were stressed. This increased stress caused the cells to produce excess amount of [reactive oxygen species](#) (ROS) which can damage cell proteins and DNA when overproduced. This destruction eventually destroys the glomerulus which in turn damages overall kidney function.

Knowing the link between reactive oxygen species and cell damage, the research team was able to measure the molecules linked to the production of the excessive ROS. These observations suggest a biomarker could be developed to signal the early development of kidney disease in humans. Researchers used an experimental small molecule to specifically block the production of mitochondrial ROS and found that mice that were destined to develop diabetic kidney disease were spared from the disorder. The study's senior investigator, Ilse S. Daehn, PhD, substantiated this hypothesis by examining urine and kidney biopsies from human patients with diabetic kidney disease. They found similar molecules suggesting stressed mitochondrial cells and increased DNA damage.

“These findings in human samples go a long way to substantiate our hypotheses which is exciting because it represents a new way forward to understanding and treating diabetic kidney disease,” Dr. Daehn says.

Continued research like this from the Icahn School of Medicine brings us one step closer to reducing the incidence of kidney disease.

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