High urea levels in chronic kidney failure might be toxic after all

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It is thought that the elevated levels of urea (the byproduct of protein breakdown that is excreted in the urine) in patients with end-stage kidney failure are not particularly toxic.

However, a team of researchers, at Albert Einstein College of Medicine, New York, and Università degli Studi di Foggia, Italy, has now generated evidence in mice that high levels of urea are indeed toxic. They therefore suggest that blocking the effects of high levels of urea might benefit patients with end-stage kidney disease.

The team, led by Michael Brownlee and Ida Giardino, found that fat cells cultured in the presence of disease-relevant levels of urea produced toxic molecules known as reactive oxygen species (ROS).

These caused the fat cells to take on characteristics of fat cells in patients with end-stage kidney disease (such as an inability to respond to the hormone insulin, which is also a key feature of type 2 diabetes). In a mouse model of end-stage kidney disease, increased ROS levels were detected, as was resistance to the effects of insulin. As treatment with an antioxidant (which will act to mop up the ROS) restored the ability of mice to respond to insulin, the authors suggest that drugs targeting ROS might help improve the quality of life and lifespan of individuals with end-stage kidney disease.

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