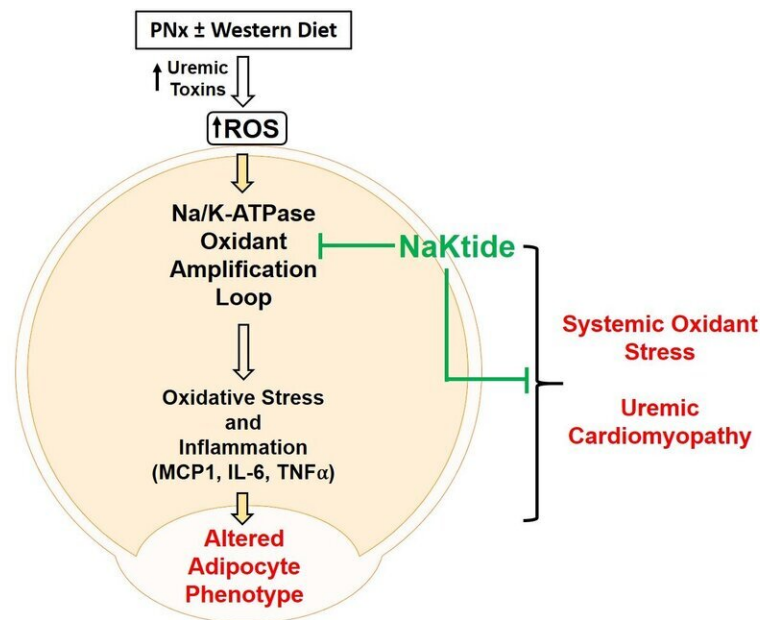


# Fat cells found to play a central role in renal failure-associated cardiomyopathy

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**Graphical Abstract:** In an experimental model of uremic cardiomyopathy in the mouse (PNx), uremic toxins induce increased production of ROS within adipocytes. This can be exacerbated by a western diet (high in fat and fructose). As ROS stimulate further generation of ROS through a Na,K-ATPase oxidant amplification loop, we targeted this loop with adipocyte specific expression of the peptide NaKtide. ROS stress in the adipocyte appears to induce a phenotypical change resulting in increases in inflammatory cytokine production. Attenuation of this

Fat cells found to play a central role in renal failure-associated cardiomyopathy.  
Credit: Marshall University Joan C. Edwards School of Medicine

New research from a team at the Marshall University Joan C. Edwards School of Medicine reveals the central role of fat cells in the systemic oxidant stress observed in renal failure-associated cardiomyopathy.

The research, published June 25 in the *Journal of the American Society of Nephrology*, is the first publication to demonstrate such an important role for [fat cells](#) known as adipocytes in a disease previously thought to have little involvement of such tissues.

Using a mouse model of experimental [renal failure](#) and a diet enriched in fat and fructose to simulate a western diet, the researchers found that production of the peptide NaKtide in fat cells inhibited the signaling function of the sodium pump, Na/K-ATPase. The peptide also prevented the development of renal failure-associated cardiomyopathy as well as other consequences of renal failure such as anemia. Targeting NaKtide production to skeletal muscle cells with a similar manipulation had essentially no effect on the cardiomyopathy or anemia in mice with experimental renal failure.

"This research provides an important breakthrough with translational application and demonstrates that Na/K-ATPase oxidant-amplification loop and/or adipocytes are potential targets for disease intervention," said lead author Komal Sodhi, M.D., associate professor of surgery and [biomedical sciences](#) at the Marshall University Joan C. Edwards School of Medicine.

Future research will help determine if these findings can be confirmed in humans, representing a novel and successful therapeutic target in [chronic renal failure](#).

"According to this novel study, targeting this oxidant amplification loop in adipocytes could serve as a viable clinical strategy for the prevention and treatment of renal failure-associated [cardiomyopathy](#)," said Joseph I. Shapiro, M.D., dean of the Joan C. Edwards School of Medicine and the study's senior author.

**More information:** Komal Sodhi et al, Central Role for Adipocyte

Na,K-ATPase Oxidant Amplification Loop in the Pathogenesis of Experimental Uremic Cardiomyopathy, *Journal of the American Society of Nephrology* (2020). [DOI: 10.1681/ASN.2019101070](https://doi.org/10.1681/ASN.2019101070)

Provided by Marshall University

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