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# Prolonged high fat diet induces kidney injury by decreased hydrogen sulfide and increased oxidative stress in mice

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**Objectives:** Obesity is an independent risk factor for the development of chronic kidney disease (CKD). Hydrogen sulfide ($H_2S$), an endogenous gaseous signaling molecule, has various biological functions, including the regulation of oxidative stress and reactive oxygen species (ROS). Recently, it has been reported that patients with obesity present low level of $H_2S$. However, the role of $H_2S$ and its producing enzymes on obesity-related kidney dysfunction remain to be defined. Here, we investigated the role of $H_2S$ in high-fat diet-induced kidney dysfunction and its association with oxidative stress in mice.

**Methods:** Nine-week old male mice were fed with either high-fat diet (HFD) or normal-fat diet (NFD) for 16 weeks. Kidney functions and structure were assessed by creatinine clearance (CrCl) and kidney tubular cell damage. $H_2S$, glutathione (GSH), and oxidative stress levels and apoptosis were measured in kidneys.

**Results:** Renal functions as monitored by CrCl and plasma creatinine (PCr) concentration declined in HFD mice. HFD feeding decreased $H_2S$ levels and $H_2S$ producing enzymes (cystathione γ-lyase, cystathionine β-synthase, and 3-mercaptopyruvate sulfurtransferase) in the kidneys. HFD-feeding increased GSSG/(GSH + GSSG) ratio, $H_2O_2$ levels, and 4-HNE, an index of lipid peroxidation, expression in the kidneys. In addition, renal tubular cell apoptosis was greater in the HFD mice than NFD mice.

**Conclusions:** These data show that HFD reduced activity of $H_2S$ producing system and increased oxidative stress leading to kidney dysfunctions, suggesting that obesity-induced kidney dysfunction is associated with the impairments of $H_2S$-producing system.