Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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PPAR-alpha Activation Alleviates Obesity-Exacerbated Renal Ischemia/Reperfusion Injury by Inhibiting Ferroptosis

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Objectives : Renal ischemia/reperfusion injury (IRI) is one of the most significant causes of acute kidney injury (AKI), commonly occurring in heart failure, ischemic shock, kidney transplantation, and partial nephrectomy, and significantly impacts the renal function. Evidence suggests that obesity may exacerbate IRI-induced AKI (IRI-AKI), while the mechanisms remain unclear. Recent studies indicate that the interplay between lipid metabolism and ferroptosis may play a critical role in obesity-related diseases. The primary aim of this study was to investigate the impact of obesity on renal IRI and to elucidate the underlying molecular mechanisms through which obesity exacerbates IRI-AKI via the ferroptosis pathway.
Methods : A diet-induced obesity (DIO) mouse model was established to simulate obesity's effects on renal IRI. High-throughput RNA sequencing analyzed gene expression changes, focusing on ferroptosis. DIO mice were treated with the PPARα agonist fenofibrate to assess effects on IRI severity and ferroptosis. A palmitic acid (PA)-induced cellular ferroptosis model was used to directly examine the role of PPARα, with CUT&Tag technology and dual-luciferase reporter assays employed to study its regulation of GPX4 expression
Results : RNA sequencing analysis revealed significant activation of the ferroptosis pathway in DIO mice subjected to IRI. Treatment experiments demonstrated that fenofibrate significantly attenuated IRI in DIO mice and effectively reduced the occurrence of ferroptosis. Similarly, in the PA-induced cellular ferroptosis model, fenofibrate conferred substantial protection, highlighting its potential therapeutic value in modulating ferroptosis. Mechanistic studies using CUT&Tag and luciferase reporter assays confirmed that PPARα directly regulates the expression of GPX4.
Conclusions : By improving lipid metabolism and suppressing ferroptosis, PPARα agonists such as fenofibrate could offer new avenues for treating obesity-related renal injury. These results provide theoretical insights and identify a potential target for the clinical management of obesity-associated AKI.

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