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Intermittent Fasting Mitigates Kidney Fibrosis Following Ischemia-Reperfusion Injury by Suppressing Cellular Senescence

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Objectives : Cellular senescence is a key contributor to maladaptive kidney repair, promoting fibrosis after acute kidney injury (AKI). Intermittent fasting (IF) has been reported to counteract cellular senescence across multiple tissues. In this study, we explore whether IF can mitigate kidney fibrosis by inhibiting cellular senescence in a mouse model of ischemia-reperfusion injury (IRI)-induced AKI.
Methods : Male C57BL/6 mice were subjected to unilateral IRI to induce AKI. Following injury, mice in the IF group adhered to a 5:2 fasting protocol, abstaining from food for 24 hours on two non-consecutive days per week over a two-month period. The experiment included four groups: control, IF, IRI, and IF+IRI.
Results : Two months post-injury, the IRI group exhibited marked kidney fibrosis, whereas fibrosis was significantly reduced in the IF + IRI group. Furthermore, the IF + IRI group demonstrated lower protein expression of p21 and decreased mRNA levels of Cdkn1a and Cdkn2a compared to the IRI group. Expression of key senescence-associated secretory phenotype (SASP) markers, including TNF-α, TGF-β, IL-6, IL-1α, and IL-1β, was also significantly downregulated in the IF + UIRI group. Additionally, IF upregulated metabolic regulators linked to energy homeostasis, such as SIRT1, p-AMPK, and PGC-1α, in the IF + IRI group compared to the IRI group.
Conclusions : These findings indicate that IF exerts protective effects against kidney fibrosis following IRI, potentially through the suppression of cellular senescence.