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FGF2-FGFR Signaling Promotes Maladaptive Repair in Folic Acid-induced Acute Kidney Injury Mouse Model

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Objectives : Many epidemiological studies revealed that survivors from the episode of AKI exhibit a persistently increased risk of progression to CKD. It has been hypothesized that the transition of AKI to CKD may result in an uncontrolled and unbalanced tissue repair process leading to renal fibrosis. The fibroblast growth factors (FGFs)-FGFR axis may play a role not only in kidney development but also in controlling the progression of AKI.
Methods : In the current study, we applied an ATP-competitive, selective FGFR kinase inhibitor, BGJ-398 (BGJ), and recombinant FGF2 and FGF9 to elucidate the regulatory role of the FGFs-FGFR axis in the recovery process in a Folic acid (FA)-induced AKI mouse model.
Results : A single dose of FA induced characteristic renal impairment by day 5, evidenced by weight loss, hydronephrosis, and increased serological markers (BUN, Kim-1, and NGAL). These effects were severely intensified by the addition of BGJ, which also led to early mortality in 40% within 2-3 days. Histologically, the BGJ co-treatment group displayed more extensive renal damage, with tubular dilation and atrophy radiating from the cortex to the medulla, than the FA-alone group. Co-administration of BGJ also significantly altered the transcriptome, specifically by up-regulating Havcr1 and Lcn2 and down-regulating Egf mRNA and was accompanied by elevated protein levels of PCNA and alpha-SMA. The treatments with rFGF2 and rFGF9 have shown distinct profiles. rFGF9 showed the potential ability to ameliorate these pathological changes, restoring both systemic and renal parameters toward baseline. In contrast, while rFGF2 partially mitigated the initial weight loss, it failed to improve other clinical or biochemical indicators of AKI and further amplified the protein expression of PCNA and alpha-SMA.
Conclusions : While the specific regulatory roles of different FGF family members remain to be fully elucidated, the current study demonstrates that FGF2-FGFR signaling drives maladaptive repair following AKI.