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Rac1 inhibition mitigates ischemia/reperfusion-induced renal injury

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Objectives: Cell migration is well recognized as a critical component of the inflammatory response and plays an important role in renal ischemia/reperfusion (I/R) injury. Rac1, a member of the Rho-like GTPases, is a key regulator of cell migration. Therefore, we investigated the role of Rac1 on the macrophage migration and kidney injury after kidney I/R.

Methods: Mice were intraperitoneally injected with either NSC23766, an inhibitor of Rac1, or vehicle for 3 days daily and then subjected to either 30 minutes of bilateral ischemia or sham operation.

Results: Kidney functional and morphological impairments were significantly increased after I/R. This I/R insult increased the Rac1 expression 24 h later and this increase were sustained until 21 days later. After I/R, F4/80-positive cells, monocytes/macrophages, increased in the interstitium of kidney. Most of the F4/80-positive cells expressed Rac1. Treatment with NSC23766 suppressed I/R-induced functional and morphological kidney impairments, along with a decrease in macrophage infiltration. Treatment of MCP-1, a chemoattractant cytokine, increased the migration of Raw 264.7 cells, a monocyte and macrophage, and this migration was inhibited by NSC23766 treatment.

Conclusions: These results indicate that Rac1 inhibition attenuates I/R-induced kidney injury by suppressing macrophage infiltration into injured sites, suggesting that Rac1 is a potentially useful target for kidney I/R injury.