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Cyanidin-3-glucoside inhibits ferroptosis in the profression of AKI to CKD

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Objectives : Renal fibrosis is a significant pathogenesis of chronic kidney disease (CKD) and ferroptosis exerts a pivotal role in CKD progression. Cyanidin-3-glucoside (C3G), a typical flavonoid that has anti-inflammatory and antioxidant effects. However, whether C3G can ameliorate ferroptosis in CKD is not known.
Methods : Hypoxia/reoxygenation (H/R)-induced HK-2 cells and I/R-AKI mice were treated with C3G with or without inhibiting AMPK. The level of intracellular free iron, the expression of the ferroptosis-related proteins acyl-CoA synthetase long chain family member 4 (ACSL4) and glutathione peroxidase 4 (GPX4), and the levels of the lipid peroxidation markers 4-hydroxynonenal (4-HNE), lipid reactive oxygen species (ROS) and malondialdehyde (MDA) were examined.
Results : We observed the inhibitory effect of C3G on ferroptosis in vitro and in vivo, which was characterized by the reversion of excessive intracellular free iron accumulation, a decrease in 4-HNE, lipid ROS, MDA levels and ACSL4 expression, and an increase in GPX4 expression and glutathione (GSH) levels. Notably, the inhibition of AMPK by CC significantly abrogated the nephroprotective effect of C3G on I/R-AKI models in vivo and in vitro.
Conclusions : Our results provide new insight into the nephroprotective effect of C3G on acute I/R-AKI by inhibiting ferroptosis by activating the AMPK pathway.