Baicalin attenuates in vivo and in vitro hyperglycemia-exacerbated ischemia/reperfusion injury by regulating mitochondrial function in a manner dependent on AMPK.

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Abstract

Cerebral ischemia/reperfusion (I/R) is a lethal and disabling disease. Studies have suggested that hyperglycemia is a risk factor for cerebral I/R. Baicalin is a natural bioactive flavonoid extracted from Scutellaria baicalensis Georgi with neuroprotective activity. In the present study, we investigated the effects of baicalin on hyperglycemia-exacerbated cerebral I/R injury. Streptozotocin (STZ) injection aggravated the brain damage induced by middle cerebral artery occlusion (MCAO) surgery, while baicalin administration reduced blood glucose, relieved neurological deficit and decreased infarct volume. In vitro, Oxygen-glucose deprivation/ reperfusion (OGD/REP) induced inordinate reactive oxygen species (ROS) production and mitochondrial dynamic impairments were markedly increased under high glucose (HG) condition. Baicalin treatment in PC12 cells inhibited dynamin-related protein 1 (Drp-1) expression, decreased mitochondrial fission, promoted mitofusin-2 (MFN2) generation, increased Drp-1 Ser637 phosphorylation, and elevated mitochondrial membrane potential ($\Delta\psi m$) via the suppression of ROS production. However, AMPKα1 knockdown abolished the protective effects of baicalin. Baicalin also suppressed cell apoptosis and enhanced mitophagy. These results suggested that baicalin protected against hyperglycemia aggravated I/R injury by regulating mitochondrial functions in a manner dependent on AMPK.