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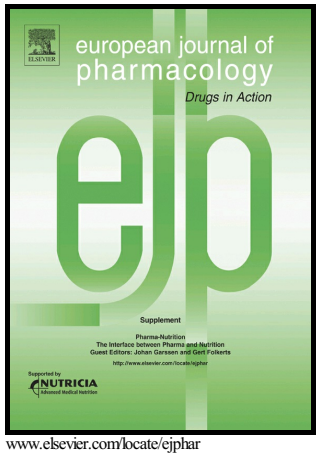

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Ginkgolide K promotes astrocyte proliferation and migration after oxygen-glucose deprivation via inducing protective autophagy through the AMPK/mTOR/ULK1 signaling pathway

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ABSTRACT

Ischemic stroke is the leading cause of death around the world. Ginkgolide K (GK) has been used to treat ischemic stroke due to its neuroprotective potential. However, the molecular mechanism underlying the neuroprotective effect of GK in ischemic stroke is still almost blank. In this study, astrocytes were divided into four groups: control group, oxygen-glucose deprivation (OGD) group, OGD + GK group and OGD + GK + Compound C (CC) group. The viability and proliferation of astrocytes were examined by Cell Counting Kit-8 assay and 5-ethynyl-20-deoxyuridine (EdU) assay, respectively. Transwell migration and wound scratch assays were conducted to evaluate astrocyte migration. The protein expression in astrocytes were determined by western blot assay. We found that GK pretreatment promoted astrocyte proliferation and migration after OGD as shown by the increase in the viability of astrocytes, glial fibrillary acidic protein level, the number of EdU positive cells and migrated cells, and the migration distance. GK pretreatment induced autophagy after OGD, as indicated by upregulation of autophagy-related protein 7, Beclin-1 protein and

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