Calenduloside E

Calenduloside E (CE) isolated from Aralia elata (Miq.) Seem. is a natural triterpenoid saponin that can reportedly ameliorate myocardial ischemia/reperfusion injury. However, its potential roles and mechanism in Cerebral ischemia-reperfusion injury are barely understood. Li et al. established an oxygen-glucose deprivation/reoxygenation (OGD/R) model in HT22 cells. They found that CE significantly attenuated the OGD/R-induced inhibition of cell viability and apoptotic cell death in HT22 cells. Moreover, CE treatment significantly ameliorated OGD/R-induced mitochondrial fission by inhibiting mitochondrial dynamin-related protein 1 (Drp1) recruitment and increasing Drp1 phosphorylation at Ser637. CE treatment significantly ameliorated OGD/R-induced mitochondrial dysfunction by increasing the mitochondrial membrane potential and reducing the mitochondrial ROS and cellular calcium accumulation. Moreover, CE treatment significantly inhibited the OGD/R-induced release of mitochondrial Cytochrome C and increase Bax, Cleaved-caspase3, and Cleaved-caspase9 protein levels, whereas CE treatment significantly reversed the OGD/R-induced decrease in Bcl-2 and full length of caspase3 and caspase9 protein levels. In vivo, we found that CE treatment significantly ameliorated ischemic/hypoxia-induced brain infarct volume, neurological deficits, and neuronal apoptosis in mice after middle cerebral artery occlusion and reperfusion. CE treatment also significantly ameliorated the mitochondrial transmembrane potential, decreased Cytochrome C release, and reversed the increase in Bax, Cleaved-caspase3, and Cleaved-caspase9 protein levels and the decrease in Bcl-2 and full length of caspase3 and caspase9 protein levels induced by cerebral ischemia/reperfusion (I/R). All these results indicated that CE treatment exerted a neuroprotective effect by ameliorating mitochondrial dysfunction during cerebral I/R injury [1)].

1)
Li J, Bu Y, Li B, Zhang H, Guo J, Hu J, Zhang Y. Calenduloside E alleviates cerebral ischemia/reperfusion injury by preserving mitochondrial function. J Mol Histol. 2022 Jul 12. doi: 10.1007/s10735-022-10087-5. Epub ahead of print. PMID: 35819738.