허혈−재관류 유도 SH−SY5Y 모델에서 미토콘드리아 매개 Apoptosis 기전 제어를 통한 초석잠 추출물의 세포보호 효과

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Stachys sieboldii Miq. Protects SH-SY5Y Cells Against Oxygen-Glucose Deprivation/Reoxygenation-Induced Injury by Inhibition of Mitochondrion-Mediated Apoptosis Pathway

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Oxygen glucose deprivation/re-oxygenation (OGD/R) induces neuronal injury via mechanisms that are believed to mimic the pathways associated with brain ischemia. *Stachys sieboldii* Miq. (chinese artichoke), which has been extensively used in oriental traditional medicine to treat of ischemic stroke; however, the role of *S. sieboldii* Miq. (SSM) in OGD/R induced neuronal injury is not yet fully understood. The present research is aimed to investigate the protective effect and possible mechanisms of SSM extract treatment in an in vitro model of OGD/R to simulate ischemia/reperfusion Injury. Pretreatment of these cells with SSM significantly attenuated OGD/R-induced production of reactive oxygen species (ROS) by increasing GPx, SOD, and decreasing MDA. SSM decreased mitochondrial damage caused by OGD/R injury and inhibited the release of cyt-c from mitochondrion to cytoplasm in SH-SY5Y cells. Furthermore, neuronal cell apoptosis caused by OGD/R injury was inhibited by SSM, and SSM could decrease apoptosis by increasing ratio of Bcl-2/Bax and inhibiting caspase signaling pathway in SH-SY5Y cells. SSM demonstrated a neuroprotective effect on the simulated cerebral ischemia in vitro model, and this effect was the inhibition of mitochondria-mediated apoptosis pathway by scavenging of ROS generation. Therefore, SSM may be a promising neuroprotective strategy against ischemic stroke.

Key words: *Stachys sieboldii* Miq., Oxygen glucose deprivation/reoxygenation, Mitochondrial damage, Reactive oxygen species, Apoptosis

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