The suppression effects of Ratanasampil on oxidative stress-induced neuronal damage and microglia-mediated neuroinflammation

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Generation of reactive oxygen species (ROS) causes lipids, proteins and DNA damage, resulting in neuronal damage and neuroinflammation. Ratanasampil (RNSP), a traditional Tibetan medicine, clinically used for the mild-to moderate AD patients living at high altitude. In vivo, RNSP improved the learning and memory in an AD mouse model (Tg2576). However, mechanism underlying the effects of RNSP is unknown. In SH-SY5Y cells, RNSP significantly ameliorated the H₂O₂-induced cytotoxicity. Furthermore, RNSP significantly reduced the H₂O₂-induced 8-oxo-2'-deoxyguanosine and attenuated the phosphorylation of p38 and ERK 1/2. In MG6 microglia, RNSP significantly ameliorated the cytotoxicity induced by hypoxia-reoxygenation. Furthermore, RNSP significantly suppressed the H6/R24-induced pro-inflammatory cytokines, ROS, DNA damage and phosphorylation of I κ B α . These observations suggest that RNSP suppressed the H₂O₂-induced neuronal death through downregulation of p38-ERK activation and regulated the H/R-induced neuroinflammation through inhibition of oxidative stress and the activation of NF κ B in MG6 cells. Therefore, RNSP may be beneficial for preventing oxidative stress-induced neuronal death and neuroinflammation.