P02-312

PROTECTIVE EFFECT OF NIGELLA SATIVA EXTRACT AND THYMOQUINONE ON SERUM/GLUCOSE DEPRIVATION-INDUCED PC12 CELLS DEATH

H.R. Sadeghnia[1,2,3], S.H. Mousavi[1,4], Z. Tayarani-Najaran[1], M. Asghari[5]
[1]Pharmacology, [2]New Sciences and Technologies, [3]Neuroscience Research Center (NRC), [4]Pharmacological Research Center of Medicinal Plants, Mashhad University of Medical Science (MUMS), [5]Biochemistry, Payame-Noor University, Mashhad, Iran

The serum/glucose deprivation (SGD)-induced cell death in cultured PC12 cells represents a useful in vitro model for the study of brain ischemia and neurodegenerative disorders. Nigella sativa L. and its active component, thymoquinone (TQ) have been known as a source of antioxidants. In the present study, the protective effects of N. sativa and TQ on cell viability and reactive oxygen species (ROS) production in cultured PC12 cells were investigated under SGD conditions. PC12 Cells were pretreated with different concentrations of N. sativa extract (15.62-250 µg/ml) and TQ (1.17-150 µM) for 2 h and then subjected to SGD for 6 or 18 h. Cell viability was quantitated by MTT assay. Intracellular ROS production was measured by flow cytometry using 2´,7´-dichlorofluorescin diacetate (DCF-DA) as a probe. SGD induced significant cells toxicity after 6, 18, or 24 h ($p< 0.001$). Pretreatment with N. sativa (15.62-250 µg/ml) and TQ (1.17-37.5 µM) reduced SGD-induced cytotoxicity in PC12 cells after 6 and 18 h. A significant increase in intracellular ROS production was seen following SGD ($p< 0.001$). N. sativa (250 µg/ml, $p< 0.01$) and TQ (2.34, 4.68, 9.37 µM, $p< 0.01$) pretreatment reversed the increased ROS production following ischemic insult. The experimental results suggest that N. sativa extract and TQ protects the PC12 cells against SGD-induced cytotoxicity via antioxidant mechanisms. Our findings might raise the possibility of potential therapeutic application of N. sativa extract and TQ for managing cerebral ischemic and neurodegenerative disorders.

https://doi.org/10.1016/S0924-9338(11)72613-X Published online by Cambridge University Press