FERULIC ACID ATTENUATES THE ISCHEMIC BRAIN INJURY-INDUCED DECREASE OF PARVALBUMIN EXPRESSION

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Ferulic acid exerts a neuroprotective effect through anti-oxidant and anti-inflammation. Parvalbumin has calcium buffering capacity and protect neuronal cells from cytotoxic Ca2+ overload. In this study, we investigated whether ferulic acid regulates parvalbumin expression in cerebral ischemia and glutamate toxicity-induced neuronal cell death. Male Sprague-Dawley rats were immediately treated with vehicle or ferulic acid (100 mg/kg, i.v.) after middle cerebral artery occlusion (MCAO) and cerebral cortex tissues were collected 24 h after MCAO. A proteomic approach elucidated the decrease of parvalbumin expression in MCAO-operated animals, ferulic acid treatment attenuated injury-induced decrease in parvalbumin expression. Moreover, RT-PCR and Western blot analyses clearly showed that ferulic acid treatment prevents the injury-induced decrease in parvalbumin levels. The numbers of parvalbumin-positive cells also decreased in MCAO-operated animals, ferulic acid attenuated the injury-induced decrease in parvalbumin-positive cells. In cultured hippocampal cells, glutamate toxicity significantly increased intracellular Ca2+ concentration, whereas increase of Ca2+ levels inhibited by ferulic acid treatment. In addition, ferulic acid treatment attenuated the glutamate exposure-induced decrease in parvalbumin levels. In conclusion, these results suggest that the maintenance of parvalbumin levels by ferulic acid in ischemic injury mediates intracellular Ca2+ levels and contributes to the neuroprotective effect of ferulic acid.