FERULIC ACID ATTENUATES THE INJURY-INDUCED DECREASE OF PROTEIN PHOSPHATASE 2A SUBUNIT B IN ISCHEMIC BRAIN INJURY

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Ferulic acid provides a neuroprotective effect during cerebral ischemia through its anti-oxidant function. Protein phosphatase 2A (PP2A) is a serine and threonine phosphatase that contributes broadly to normal brain function. This study investigated whether ferulic acid regulates PP2A subunit B in a middle cerebral artery occlusion (MCAO) animal model and glutamate toxicity-induced neuronal cell death. MCAO was surgically induced to yield permanent cerebral ischemic injury in rats. The rats were treated with either vehicle or ferulic acid (100 mg/kg, i.v.) immediately after MCAO, and cerebral cortex tissues were collected 24 h after MCAO. Ferulic acid significantly reduced the MCAO-induced infarct volume of the cerebral cortex. A proteomics approach elucidated the reduction of PP2A subunit B in MCAO-induced animals, and ferulic acid treatment prevented the injury-induced reduction in PP2A subunit B levels. RT-PCR and Western blot analyses also showed that ferulic acid treatment attenuates the injury-induced decrease in PP2A subunit B levels. Moreover, the number of PP2A subunit B-positive cells was reduced in MCAO-induced animals, and ferulic acid prevented these decreases. In cultured neuronal cells, ferulic acid treatment protected cells against glutamate toxicity and prevented the glutamate-induced decrease in PP2A subunit B. These results suggest that the maintenance of PP2A subunit B by ferulic acid in ischemic brain injury plays an important role for the neuroprotective function of ferulic acid.