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Identification of protective role of enolase1 in cerebral ischemia-induced neuronal injury and of potential ischemia biomarker by brain slice-based selex

Stroke is one of leading causes of disability and death among adults worldwide and results in numerous biochemical alterations. While the mechanisms underlying neuronal death and dysfunction remain poorly understood, we investigated the differential proteomic profiles of mouse brain homogenate with 3h of Middle Cerebral Artery Occlusion (MCAO) ischemia, or sham, by mass spectrometry. We identified Enolase1 (ENO1), a key glycolytic enzyme, as a potential mediator of neuronal injury in MCAO ischemic model. Immunohistochemical analysis revealed that ENO1 is localized in neuronal cytoplasm and dendrites. Interestingly, the expression level of ENO1 was significantly increased in the early stage, but dramatically decreased in the late stage, of cerebral ischemia *in vivo*, and of cultured hippocampal neurons treated with oxygen/glucose deprivation (OGD) *in vitro*. Strikingly, ENO1 overexpression in cultured neurons alleviated dendritic and spinal loss caused by OGD treatment. The neuronal injury caused by OGD treatment *in vitro* or ischemia *in vivo* was mitigated by the application of PEP. Taken together, these data revealed that ENO1 plays a novel and protective role in cerebral ischemia-induced neuronal injury, highlighting a potential of ENO1 as a therapeutic target of neuronal protection from cerebral ischemia. Moreover, we also utilized frozen brain slices of Middle Cerebral Artery Occlusion (MCAO) in a mouse model of ischemia to select a specific binding aptamer, termed LCW17, by tissue-based SELEX. We identified the binding target of LCW17 as Vigilin. Vigilin is increased in ischemia brain slices and exhibits enhanced release from cultured hippocampal neurons after oxygen glucose deprivation *in vitro*. In summary, Aptamer LCW17 and Vigilin, may potentially be applied to define the molecular mechanism underlying ischemic stroke, as well as its diagnosis.

Biography

Haijun Tu is a Professor, Doctoral tutor and Assistant Dean of neuroscience. He is the director of the National Demonstration International Science and Technology Cooperation Base for Biomedical and Life Analytical Chemistry. He is also the Director of Hunan University and Jinxiang Pharmaceutical Joint R&D Center. He was appointed as a professor of genetics and neuroscience at Hunan University in July 2015, and a doctoral tutor. From January 2011 to June 2015, he successively studied at the French Institute of Health and Medicine (Ecole Normale Supérieure, Paris/INSERM, U1024) and the French National Scientific Research Center/Claude Bernard Lyon First University.

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