A therapeutic hypothesis for glucose intolerance after cerebral ischemia

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Interestingly, a recent study found that ischemic stress causes hyperglycemia and may worsen ischemic neuronal damage. In addition, decreased insulin sensitivity after ischemic stress seems to be involved in the development of post-ischemic glucose intolerance. However, the involvement of brain-derived neurotrophic factor in the development of glucose intolerance following ischemic stress still remains unclear.

Xiaoliang Shu and colleagues from the Affiliated Dongfang Hospital of Tongji University, China found that after cerebral ischemia, the expression levels of brain-derived neurotrophic factor and tyrosine kinase B receptor were significantly decreased in the hypothalamus. However, intrahypothalamic administration of brain-derived neurotrophic factor suppressed the decrease in insulin receptor and tyrosine-phosphorylated insulin receptor expression in the liver and skeletal muscle, and inhibited the overexpression of gluconeogenesis-associated phosphoenolpyruvate carboxykinase and glucose-6-phosphatase in the liver of cerebral ischemic mice. In addition, serum insulin levels remained unchanged. The researchers believed that glucose intolerance after cerebral ischemia can be improved by suppressing the loss of brain-derived neurotrophic factor in the hypothalamus.

These findings were published in the *Neural Regeneration Research* (Vol. 8, No. 25, 2013).

More information: Shu XL, Zhang YS, Xu H, Kang K, Cai DL. Brain-

derived neurotrophic factor inhibits glucose intolerance after cerebral ischemia. Neural Regen Res. 2013;8(25):2370-2378.

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