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Short Communication

The Study of Neuroprotective Effects of Adipokines and Physical Exercise against Cerebral Ischemia/Reperfusion (Stroke) Injuries

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A stroke is the second leading cause of death worldwide and is the major cause of morbidity, particularly in the middle aged and elderly population that also called brain attack, Cerebrovascular Accident (CVA). It is a medical emergency that can happen to anyone at any time. It occurs when blood flow to an area of brain is cut off. When this happens, brain cells are deprived of oxygen and begin to die.

There are two kinds of stroke. The more common kind, accounting for around 85% of strokes, called ischemic stroke, is caused by a blood clot that blocks or plugs a blood vessel in the brain. The other kind, called hemorrhagic stroke, is caused by a blood vessel that breaks and bleeds into the brain. "Mini-strokes" or Transient Ischemic Attacks (TIAs), occur when the blood supply to the brain is briefly interrupted.

How a person is affected by their stroke depends on where the stroke occurs in the brain and how much the brain is damaged. For example, someone who had a small stroke may only have minor problems such as temporary weakness of an arm or leg. People who have larger strokes may be permanently paralyzed on one side of their body or lose their ability to speak. Some people recover completely from strokes, but more than 2/3 of survivors will have some type of disability.

Reduction or cessation of blood flow to a part of the brain and blockage of brain feeding leads to the transient focal cerebral ischemia. During cerebral ischemia, cerebral blood flow as well as oxygen and metabolite levels reduce, then the reperfusion leads to the return of oxygen to the cells which exhibits superoxide radicals' generation. It affects the cell signaling and ends in tissue damage. Cerebral ischemia leads to a cascade of events that causes some important cellular changes. Ischemia leads to selective loss of vulnerable neurons by apoptosis in specific brain regions. Cerebral ischemia causes tissue damage through the interaction of complex pathophysiological processes, including excitotoxicity, inflammation, and apoptosis. Furthermore, reperfusion generates an overproduction of Reactive Oxygen Species (ROS), or better known as free radicals, leading to reperfusion injury. The ultimate result of ischemic cascade initiated by acute stroke is neuronal death along with an irreversible loss of neuronal function. Therapeutic strategies in stroke have been developed with two main aims: restoration of cerebral flow and the minimization of the deleterious effects of ischemia on neurons.

Adipokines, hormones released by adipose tissue. The certain adipokines (for example, apelin and visfatin) participated in pathomechanisms of ischemic stroke.

Visfatin, a novel adipokine, is predominantly produced by visceral adipose tissue and it has been linked to a diverse variety of cellular processes and it is an important factor in cell apoptosis and survival. Our studies have indicated that visfatin entails neuroprotective effects against ischemia injury when used at the time of cerebral reperfusion. These neuroprotective mechanisms of visfatin occur through decrease the expression of proapoptotic proteins (cleaved caspase-3 and Bax) and, on the other hand, increase the expression of antiapoptotic proteins (Bcl-2). Also, other protective effects of visfatin might be related to various mechanisms including: activation of PI3K and MEK1/2, inhibition of mPTP opening, trigger a redox adaptation response, inhibition of lipid peroxidation. Thus, our findings indicate that visfatin is a new therapeutic target for cerebral ischemia.

The adipocytokine Apelin is a peptide that was isolated from a bovine stomach for the first time. This peptide and its receptor are abundantly expressed in the nervous and cardiovascular systems. According to previous studies, Apelin-13 protects cardiomyocytes from ischemic injury and apoptosis. In addition, this peptide has neuroprotective effect on hippocampal and cultured mouse cortical neurons against NMDA receptor-mediated excitotoxicity as well as cortical neurons from ischemic injury. Our findings demonstrate that treatment by Apelin-13 exerts its protective effects in ischemic models *via* blocking programmed cell-death. We suggest that Apelin-13 might be a promising therapeutic target for stroke, although more researches are necessary to take into account the potential therapeutic effects of Apelin-13 in stroke patients.

The evidence indicates that physical training has a neuroprotective role against ischemic injury. Our study demonstrated that pre-ischemic exercise training reduces hippocampal injuries after cerebral ischemia through preventing neuronal necrosis and apoptosis. In addition, exercise preconditioning ameliorated ischemia-induced memory dysfunction. Physical exercise can exert protective effects in brain ischemia models *via* some possible mechanisms such as preventing NMDA receptor cytotoxicity, reducing ROS production and upregulating ERK1/2 and HSP-70. The neuroprotective mechanisms of exercise can provide a neuroprotective therapy that will simultaneously promote cell survival and decrease neuronal death, therefore ameliorating much of the functional and memory loss following ischemic stroke.

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