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Leptin improves the locomotion recovery of spinal cord injury

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Spinal cord injury (SCI) causes long-term disability and has no effective clinically treatment. The initial trauma always results in permanent functional impairment and severe disability followed by secondary injury mechanism, which is characterized by increased inflammation, glial scarring and neuronal cell death. Leptin (a glycoprotein) could induce the activation of Janus kinase (JAK2)/signal transducers and activators of transcription-3 (Stat3) pathway via leptin receptor. In vivo, we discovered the intraperitoneal injection of leptin improved the locomotion recovery of spinal cord injury. Then, we researched the neuro-protective and anti-inflammatory role of leptin on the spinal cord neurons and astrocytes. In the cultured neurons, we discovered leptin administration could enhance the expression of caveolin-1, block the composition of P2X7R-Panx1 complex and reduce the damage to neurons induced by ATP or by modeling operation of SCI. Even without injury operation, the pretreatment with leptin could suppress neuronal Ca²⁺ imaging triggered by ATP in spinal cord of live transgenic mice. In the cultured astrocytes, we discovered that: 1) the chronic administration of leptin could suppress the release of AA and PGE2 stimulated by ATP from the cultured spinal cord astrocytes; 2) leptin could elevate the expression of caveolin-1 through JAK2/Stat3 signaling pathway; 3) the increased caveolin-1 blocked the conjunction between Src and EGFR; 4) our results highlight leptin as a promising therapeutic agent for SCI.

Biography

Maosheng Xia has completed his PhD and MD from China Medical University and two-year Post-doctoral studies from Medical Center of Rochester University School. Currently, he works as the Associate Professor and Associate Chief Physician in the First Hospital of China Medical University. He has published 7 papers in the field of spinal cord injury.

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