

Adiponectin receptor 1

The adiponectin receptors, AdipoR1 and AdipoR2, serve as receptors for globular and full-length adiponectin and mediate increased AMPK and PPAR- α ligand activities, as well as fatty acid oxidation and glucose uptake by adiponectin.

In 2016, the University of Tokyo announced it was launching an investigation into anonymously made claims of fabricated and falsified data on the identification of AdipoR1 and AdipoR2.

A study first showed that activation of adiponectin receptor 1 (AdipoR1) by AdipoRon could attenuate mitochondrial dysfunction after ICH. In vivo, experimental ICH model was established by autologous blood injection in mice. AdipoRon was injected intraperitoneally (50 mg/kg). Immunofluorescence staining were performed to explicit the location of AdipoR1, AMP-activated protein kinase (AMPK) and peroxisome proliferator-activated receptor- γ coactivator-1 α (PGC1 α). The PI staining was used to quantify neuronal survival. The expression of AdipoR1 and its downstream signaling molecules were detected by Western blotting. In vitro, 10 μ M oxyhemoglobin (OxyHb) was used to induce the neuronal injury in SH-SY5Y cells. Annexin V-FITC/PI staining was used to detect the neuronal apoptosis and necrosis. Mitochondrial membrane potential ($\Delta\psi$ m) was measured by a JC-1 kit and mitochondrial mass was quantified by mitochondrial fluorescent probe. In vivo, PI staining showed that the administration of AdipoRon could reduce neuronal death at 72 h after ICH in mice. AdipoRon treatment enhanced ATP levels and reduced ROS levels in perihematoma tissues, and increased the protein expression of AdipoR1, P-AMPK, PGC1 α , NRF1 and TFAM. In vitro, the JC-1 staining and Mito-tracker™ Green showed that AdipoRon significantly alleviated OxyHb-induced collapse of $\Delta\psi$ m and enhanced mitochondrial mass. Moreover, flow cytometry analysis indicated that the neurons treated with AdipoRon showed low necrotic and apoptotic rate. AdipoRon alleviates mitochondrial dysfunction after intracerebral hemorrhage via the AdipoR1-AMPK-PGC1 α pathway ¹⁾.

Unclassified

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