Exercise for Stroke

Exercise preconditioning has been widely accepted as a being of safe and effective preventive measure for stroke.

Pre-stroke exercise conditioning reduces neurovascular injury and improves functional outcomes after stroke. The goal of this study was to explore if post-stroke exercise conditioning (PostE) reduced brain injury and whether it was associated with the regulation of gluconeogenesis. Adult rats received 2 h of middle cerebral artery (MCA) occlusion, followed by 24 h of reperfusion. Treadmill activity was then initiated 24 h after reperfusion for PostE. The severity of the brain damage was determined by infarct volume, apoptotic cell death, and neurological deficit at one and three days after reperfusion. We measured gluconeogenesis including oxaloacetate (OAA), phosphoenolpyruvate (PEP), pyruvic acid, lactate, ROS, and glucose via ELISA, as well as the location and expression of the key enzyme phosphoenolpyruvate carboxykinase (PCK)-1/2 via immunofluorescence. We also determined upstream pathways including forkhead transcription factor (FoxO1), p-FoxO1, 3-kinase (PI3K)/Akt, and p-PI3K/Akt via Western blot. Additionally, the cytoplasmic expression of p-FoxO1 was detected by immunofluorescence. Compared to non-exercise control, PostE (*p < .05) decreased brain infarct volumes, neurological deficits, and cell death at one and three days. PostE groups (*p < .05) saw increases in OAA and decreases in PEP, pyruvic acid, lactate, ROS, glucose levels, and tissue PCKs expression on both days. PCK-1/2 expressions were also significantly (*p < .05) suppressed by the exercise setting. Additionally, phosphorylated PI3K, AKT, and FoxO1 protein expression were significantly induced by PostE at one and three days (*p < .05). In this study, PostE reduced brain injury after stroke, in association with activated PI3K/AKT/FoxO1 signaling, and inhibited gluconeogenesis. These results suggest the involvement of FoxO1 regulation of gluconeogenesis underlying post-stroke neuroprotection [1)].

The numbers of both the HSP20-containing neurons and the HSP20-containing glia inversely correlated with the outcomes of ischemic stroke. In addition, preischemic treadmill exercise improves outcomes of ischemic stroke by increasing the numbers of both the HSP20-containing neurons and the HSP20-containing glia [2)].

1)
Li F, Geng X, Ilagan R, Bai S, Chen Y, Ding Y. Exercise postconditioning reduces ischemic injury via suppression of cerebral gluconeogenesis in rats. Brain Behav. 2022 Nov 30:e2805. doi: 10.1002/brb3.2805. Epub ahead of print. PMID: 36448290.

2)
Lin CM, Chang CK, Chang CP, Hsu YC, Lin MT, Lin JW. Protecting against ischemic stroke in rats by HSP 20-mediated exercise. Eur J Clin Invest. 2015 Oct 19. doi: 10.1111/eci.12551. [Epub ahead of print] PubMed PMID: 26479875.