Abstract

Exercise therapy has been a mainstay of rehabilitation after ischemic brain damage, but the rationale of exercise therapy in molecular biology level has not been established. Previous studies have suggested that the neurotrophic factors and their receptors are involved in brain plasticity, memory and learning, and may be neuroprotective following ischemic brain damage.

This study was undertaken to determine whether exercise in ischemic brain injury could improve motor behavior index and to analyze whether exercise change the expressions of the neurotrophic factors and receptors.

The Sprague–Dawley male rats (N=35) were anesthetized and subjected to permanent occlusion of the left middle cerebral artery (MCAO). Treadmill exercise for 2 weeks is independent variable. Motor behavior index by Garcia was measured at post–infarct 2–day, 9–day and 16–day. Western blots of both hemisphere excluding brain stem and cerebellum were performed for BDNF, NT–3, NT–4, trkA, trkB, trkC at post–infarct 16–day. Immunohistochemistry of the brain was done for NGF, BDNF, NT–3, NT–4, trkA, trkB, trkC. Sham controls (N=8) were also divided into exercise and no exercise group.

The results were as follows.

1. Treadmill exercise for 2 weeks in ischemic rat model
improved greater improvement in motor behavior index, and increased expression of BDNF, NT-3, NT-4, trkA, trkB, trkC in contralateral hemisphere.

2. Treadmill exercise for 2 weeks in normal rats increased expression of BDNF, NT-3, NT-4, trkA, trkB, trkC in bilateral hemisphere, and increased immunoreactivity of trkA, trkB, trkC was noted at vascular structure.

In conclusion, treadmill exercise improved the motor behavior and altered expression of neurotrophic factors and their receptors, suggesting exercise may modulate neuro-enhancing role and a role for plasticity in the brain.

Key words: Treadmill exercise, ischemia, neurotrophin, motor behavior index, tyrosine kinase, BDNF
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