Effects of Different Protocols of Physical Exercise on Markers Mitochondrial Biogenesis in Animals Induced to Parkinson’s Disease

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Introduction: The beneficial effects of exercise on Parkinson’s disease (PD) have been suggested, but the mechanisms responsible for these effects are poorly understood. The aim of this study was to investigate the preventive effects of different exercise programs on markers of mitochondrial biogenesis in striatum and hippocampus of mice induced to PD. Material and Methods: Seventy-two male mice C57, 2-month-old, randomly divided: untrained+operated (Sham), untrained+PD (PD), trained+operated (TTR), trained strength+operated (TSG), trained+PD (PD+TTR), trained strength +PD (PD+TSG), n=12. The animals were subjected to a treadmill (8 weeks, 4 days (alternate) / 50 min / 13-17 m/min), and strength (5 times (40 sessions) in the apparatus for resistance training scale to scale. Twenty-four hours after, PD was induced by unilateral lesion in the left hemisphere with an injection of 6-OHDA (2 μg/1ul ascorbic acid 0.2%). Seven days after injury, were submitted to the open field test, forced swim test and the test of rotameter. The striatum and hippocampus homogenates in buffer specific for the Western blotting with anti-peroxisome proliferator-activated (PPAR-γ), anti-co-activator transcription-1 alpha (PGC-1 α) and activity of complex I. Results and Discussion: The PD, PD+TTR and PD+TSG groups showed increase numbers of rotation. However, exercise training was effective to reduce the numbers of rotation in PD+TTR and PD+TSG, when compared to PD group. The exercise training in mice with PD (TSG and PD+TSG) showed a significant decrease in the activity of PGC1-α, PPAR-γ and complex I in the striatum and hippocampus. There was a significant increase in PPAR-γ in the striatum and hippocampus in the PD group compared to the sham group and PD+TSG between groups. Conclusions: Our results suggest that exercise modulates the activity of markers of biogenesis as PPAR-γ, PGC1-α and complex I in striatum and hippocampus of mice, improving our understanding of the pathogenesis of PD.

Key Words: Parkinson’s Disease., Physical exercise., Mitochondrial biogenesis., Oxidative stress., 6-OHDA.

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