Running Decreases Motor Changes Associated to Dopamine D<sub>2</sub>-like Receptors Imbalance in Neurotoxicant Models of Parkinsonism

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Parkinson’s disease is the most prevalent motor neurodegenerative disease. Over the last decade, regular physical exercise has been implicated to an increased plasticity in the adult brain and it might afford protection against dopaminergic neurons death, motor and non-motor dysfunction elicited in experimental models. Male C57BL/6 mice were assigned to two groups: untrained and runners, using a moderate-intensity treadmill during 6 weeks. The animals were treated with 6-OHDA (4 μg, right midstriatum, AP 0.4, DL 1.8, DV 3.5) or MPTP (65 mg/kg, intranasal), or their respective vehicle 48 h after the end of physical program. In the striatal 6-OHDA model, apomorphine (0.6 mg/kg, s.c.) induced a progressive rotation in sedentary animals, which was not observed in the 6-OHDA exercised mice, suggesting a reduced dopamine receptors sensitization in trained mice. Moreover, MPTP-treated groups also showed dopamine receptor sensitization as indicated by a marked increase in climbing behavior induced by apomorphine (0.2 mg/kg, s.c.) that was prevented by exercise. Indeed, exercise prevented the catalepsy induced by haloperidol (0.32 mg/kg, i.p.) in MPTP-treated mice. These effects seem not be related to neuroprotective actions of exercise since it did not prevent the MPTP-induced reduction in the levels of dopamine and enzyme tyrosine hydroxylase in the striatum. However, exercised MPTP-treated mice presented decreased striatal DA turnover, at same time of increased striatal dopamine D<sub>2</sub>-like receptor density, in comparison to sedentary MPTP-treated mice. Taken together, the present findings suggest that exercise can reduce behavioral alterations associated to dopamine receptors imbalance in neurotoxicant models of Parkinsonism.

Word Keys: Parkinson’s disease; exercise; striatum; MPTP; 6-OHDA.

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