# Effect of Physical Activity upon Mitochondrial Trafficking and Degradation in a Rat Model of Parkinson's Disease

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Parkinson's disease (PD) occurs when dopaminergic neurons start to degenerate. It isn't clear what happens to initiate PD, but it's known that a mitochondrial respiratory chain dysfunction and a significant atrophy of the substantia nigra and locus coeruleus are related to PD. Considering that the practice of physical activity (PPA) has demonstrated positive effects upon PD clinically, I aim to evaluate the effect of PPA upon the expression mitochondrial trafficking and degradation proteins in cells extracted during the development of the disease. I hypothesized that PPA during the development of PD could decrease the dysfunction in mitochondrial trafficking and degradation related to the disorder. The proteins expression analyzed in cells, donated to the project, from a rat model of PD submitted to different protocols of physical activity, were: dynein, dynactin, syntaphilin, kinesin-1, and kinesin-5, and Tom-20. Proteins from the samples were separated by Western Blot, and analyzed quantitatively and qualitatively. Since there was no significant alteration, upon the existent dysfunction due to the disease, in the expression of proteins responsible for trafficking and degradation in any of the groups, I conclude that the PPA wasn't capable to minimize the alterations in the mechanisms of mitochondrial trafficking and degradation in this model, therefore the hypothesis was refuted. However, unexpectedly, the PPA alone led to alterations in protein expression, similarly to those identified with PD development. Furthermore, a lack of impact PPA in substantia nigra corroborates with previous clinical results and it's important to initiate a new research pathway in PD research.

**Awards Won:**