



EFFECTS OF CURCUMIN AND FISH OIL ON BEHAVIORAL PARAMETERS IN AN EXPERIMENTAL MODEL OF PARKINSON'S DISEASE

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INTRODUCTION

Parkinson's disease (PD) is a multifactorial neurodegenerative disease characterized by bradykinesia, muscle stiffness and loss of automatic movements. Fish oil (FO), consisting of omega-3 polyunsaturated fatty acids, plays an important role in neurodegeneration, influencing the lipid composition of plasma and mitochondrial membranes. Curcumin (CUR) is a flavonoid compound with antioxidant and neuroprotective properties, reducing the production of reactive species. Therefore, the objective of this work was to investigate the neuroprotective effects of FO, CUR and its association in mice submitted to a rotenone, that induces PD through behavioral evaluation.

MATERIAL AND METHODS

Male Swiss mice (3 months) (n= 8 animals/group) were used, kept at the Biological Laboratory of the Biological Sciences Institute of the Federal University of Rio Grande-FURG. The experimental protocol was approved by the FURG Ethics Committee on Animal Use (Nº P056/2015). The animals were treated with soybean oil (control), CUR (CUR50, 50 mg/Kg), fish oil (FO300, 300 mg/Kg) and the combination of the two compounds (CUR50+FO300) orally for 30 days. From day 8, they were exposed to rotenone (ROT, 1 mg/Kg) intraperitoneally until day 30. At the end of the treatment, the animals were submitted to behavioral tests: Open Field, Beam Walking, Foot Fault and Pole Test. Statistical analysis

was done by one-way ANOVA followed by post-hoc Newman-Keuls test.

RESULTS

The foot fault results showed that mice receiving ROT, CUR50, FO300 and CUR50 + FO300 showed a significant increase in the number of slats in the grid compared to the control group. In the beam walking test the ROT administration increased the number of slides compared to the control group, while treatments with CUR50, FO300 and CUR50+FO300 reduced the number of slides. In the open field test, the ROT and FO300 treatment significantly decreased the distance traveled, however, there were no changes in the number of animals raised. Finally, using the Pole Test, ROT administration increased animal latency, while CUR50, FO300 and CUR50+FO300 treatments prevented ROT-induced damage.

CONCLUSIONS

We observed that CUR and FO showed protection against the damage caused by rotenone on the animals' motor balance and bradykinesia. However, the association had no additional effects when compared to the isolated compounds. Thus, further analysis will be necessary to verify the mechanisms involved in the neuroprotective effect of the compounds.

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