Establishment of a new *in vitro* model of neuroinflammation associated to Parkinson's disease induced by aminochrome and characterization of neuroprotective action of apigenin.

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INTRODUCTION: The mechanisms responsible for neurodegeneration in Parkinson's disease are still unknown, however, some cellular and molecular disorders are involved in this process: accumulation of α-synuclein, oxidative stress, mitochondrial damage, proteosomal, autophagic dysfunction and neuroinflammation. Among these, only neuroinflammation has not yet been associated with effects induced by aminochrome. This neurotoxin originates from dopamine oxidation in cytosolic pH, that in specific conditions can accumulate in dopaminergic neurons and generate cytotoxicity. **OBJECTIVES:** This work is mainly focused on study of neuroinflammatory potential of aminochrome and role of apigenin a well known immunomodulator flavonoid in reversing this phenotype. **MATERIAL AND METHODS:** Midbrain organotypic cultures derived from wistar rats 8 days were cultivated for 3 days with DMEM/F12, incubated in 5% CO_2, at 37 ° C. Slices were treated with aminochrome (0,01 to 25 µM) and/or apigenin (10µM), and analyzed after 24 or 48 hours. Neurotoxicity was evaluated by morphological analysis and expression of tyrosine hydroxylase (TH) by Western blot. The neuroinflammatory response was assessed by immunohistochemistry for Iba1 and RTq-PCR for cytokines TNF and IL-1β. The modulatory effects of neurotrophic factors were evaluated by RTq-PCR. **RESULTS AND DISCUSSION:** Our results demonstrate aminochrome induced tissue damage in culture, associated with reduction of TH expression, which was inhibited by apigenin. Furthermore, aminochrome induced morphological changes in $Iba1^+$ cells, followed by increased expression of mRNA for TNF and IL-1β, which were also inhibited by apigenin. Aminochrome reduced mRNA expression of CDNF and NGF but increased BDNF. Apigenin inhibited the effects induced by aminochrome in relation to CDNF and NGF expression. On the other hand, apigenin treatment reduced expression of BDNF and GDNF. **CONCLUSION:** These results demonstrated that neuroinflammation and neurotrophic factors are also involved in the neurodegeneration induced aminochrome and that apigenin is a potential protective agent against cell damage induced by this toxin.

Keywords: Parkinson's disease, aminochrome, apigenina.

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