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The Association of Palmitoylethanolamide with Luteolin Decreases Neuroinflammation and Stimulates Autophagy in Parkinson's Disease Model.

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Abstract

Parkinson's disease (PD) is a disorder resulted by degeneration of dopaminergic neurons. To counteract the neuroinflammation and oxidative stress of PD, we decided to test a new composite constituted by palmitoylethanolamide (PEA) and luteolin (Lut), in a mass ratio of 10:1, respectively (co-ultraPEALut). In this study the neuroprotective property of the new compound was investigated. For the in vivo model of PD, mice received four injections of the dopaminergic neurotoxin 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine (MPTP). Starting 24 h after the first administration of 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), we treated animals with co-ultraPEALut daily until 7 days. On day 8, brains were processed for Western blotting and immunohistochemical analysis. Treatment with co-ultraPEALut reduced the specific markers of PD (tyrosine hydroxylase immunopositive), and the increased levels of activated astrocytes and pro-inflammatory cytokines as well as inducible nitric oxide synthase. Further, the possible association of autophagy with the beneficial effects of coultraPEALut. Western blot analysis and immunofluorescence staining showed that co-ultraPEALut administration increased autophagy process. These data were confirmed by an in vitro model, using SH-SY5Y neuroblastoma cells. Western blot analysis showed that co-ultraPEALut pre-treatment maintained high Beclin-1 and p62 expression, while continued to inhibit the p70S6K expression. Altogether, these results put forward that treatment with co-ultraPEALut is able to modulate both the neuroinflammatory process and the autophagic pathway involved in PD, actions which may underlie its neuroprotective effect.

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