Format: Abstract

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Neuroprotective Effects of Co-UltraPEALut on Secondary Inflammatory Process and Autophagy Involved in Traumatic Brain Injury.

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Abstract

Traumatic brain injury (TBI) initiates a neuroinflammatory cascade that contributes to neuronal damage and behavioral impairment. In the present study, we performed a widely used model of TBI to determine the neuroprotective propriety of palmitoylethanolamide (PEA) and the antioxidant effect of a flavonoid luteolin (Lut), given as a co-ultramicronized compound Co-ultraPEALut. We demonstrated that the treatment with Co-ultraPEALut resulted in a significant improvement of motor and cognitive recovery after controlled cortical impact, as well as markedly reducing lesion volumes. Moreover, our results revealed the ability of Co-ultraPEALut to reduce brain trauma through modulation of nuclear factor-κB activation. In addition, treatment with Co-ultraPEALut significantly enhanced the post-TBI expression of the neuroprotective neurotrophins glial cell line-derived neurotrophic factor compared with vehicle. Co-ultraPEALut at the dose of 1 mg/kg also modulated apoptosis, the release of cytokine and reactive oxygen species, the activation of chymase, tryptase, and nitrotyrosine, and inhibited autophagy. Thus, our data demonstrated that Co-ultraPEALut at a lower dose compared with PEA alone can exert neuroprotective effects and the combination of both could improve their ability to counteract the neurodegeneration and neuroinflammation induced by TBI.

KEYWORDS: autophagy; inflammation; luteolin; neuroprotection; palmitoylethanolamide

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MeSH terms, Substances

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