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PPARB/Δ ANTAGONISM RESCUES DOPAMINERGIC NEURONS IN AN IN VITRO PARKINSON'S DISEASE MODEL

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Parkinson's disease (PD) is one of the most common neurologic disorder. Motor dysfunctions are assigned as primary symptoms of PD, being all related to events starting on one side of the body. Despite a relevant number of studies, the mechanisms responsible for neurodegeneration in PD are still unknown, but oxidative stress, excitotoxicity and neuroinflammation are believed to play key roles in neuronal death. The pathogenesis of neurodegenerative diseases such as alzheimer's disease (AD) and PD has also been described as reduced neurotrophic support. There has been considerable interest in studying the involvement of neurotrophic factors, that are substances known to be crucial for the survival of specific neurochemical-phenotype classes of neuron. We have previously reported that the peroxisomal proliferator activated receptor β/δ (PPARβ/δ) is involved in the decrease of the TrkBFl in neurodegeneration. PPARs are a class of transcription factors involved in the control of several pathways both in physiological and in pathological conditions including neurodegeneration. A detrimental role for PPARβ/δ has been proposed in AD, being closely related to the decrease of BDNF and TrkBfl. On these bases, in the present work the signal transduction pathways activated in PD were dissected in two 6-OHDA in vitro models of PD (differentiated SH-SY5Y and LUHMES cells) treated with a PPARβ/δ specific antagonist. The 6-OHDA treatments determined a significant increase of neuronal death, while the presence of the antagonist rescued cell viability, thus indicating that blocking PPARβ/δ, neuronal survival pathways, such as BDNF/TrkB, p-CREB, ERK5 were restored to control conditions.

Note: