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Cotinine normalizes the morphology and abundance of astrocyte after chronic restraint

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Astrocytes maintain brain homeostasis and support neuronal function. In recent years, it has been shown a decrease in the number of astrocytes that present immunoreactivity (IR) for the fibrillary acidic protein (GFAP) in the brain of rodent models of posttraumatic stress disorder (PTSD). GFAP is a family of proteins used as a marker of astrocytes and in less extent of immature brain cells. Astroglia dysfunction seems to be involved in the development of depression and memory loss induced by stress. Cotinine, a positive modulator of the α7 nicotinic acetylcholine receptor (nAChR), prevented memory impairment, depressive-like behavior, and synaptic loss when co-administered during restraint stress. In here, we studied the effects of post-treatment with intranasal cotinine on depressive behavior, memory as well as number and morphology of GFAP+ cells, in the hippocampus and frontal cortex of chronically restrained mice. After two weeks of treatment with cotinine or vehicle, mice were tested for locomotor activity (Open Field Test), depressive-like behavior (Forced Swim test), and memory (Novel object recognition). After euthanasia, GFAP IR cells and their morphology were assessed using immunohistochemistry. This evidence revealed that in addition to the depression and cognitive impairments, restraint stress induced a significant decrease in the number of GFAP+ cells and their arborization complexity. Cotinine prevented cognitive impairment and depressive behavior and restored GFAP+ cells morphology in both brain regions. This data suggests that cotinine acts by a mechanism involving the restoration of astrocyte function after stress in mice.

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