Restoration of Neuroprotection of Glial Cells in Opioid Addiction: Case Report

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Abstract

cytokines and chemokines through activation of Toll-like receptor 4 (TLR4) and mitogen-activated protein kinase (MAPK) are implicated in opioid tolerance, known as opioid-induced hyperalgesia (OIH) . Astrocytes, exposed to prolonged stress from continuous opioid consumption, lose the ability to adequately remove excess glutamate from synapses. When combined with gamma-aminobutyric acid (GABA) inhibition, the imbalance leads to excitotoxicity and, in prolonged cases, neuronal degeneration. Such events increase pain sensitivity and reduce the neuroprotective capacity of glial cells, leaving the central nervous system vulnerable to acute extracellular changes with the potential to altered physiological and behavioral components in individuals with opioid use disorder. . Opioids have also been shown to increase the BBB activity of P-glycoprotein, a protein that increases the ux of xenobiotics across the BBB, adding another layer of complexity to nding appropriate treatments for chronic opioid use.

Behavioral and cellular mechanisms of addiction

Drugs of abuse such as opioids have the unique ability to disguise themselves as prized targets of the brain, allowing the urge to become addictive. Changes caused by addiction, a medical condition

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