Mu opioid receptor

Its a major subtype of opioid receptor

mu (μ) MOR OP3 (I) μ1, μ2, μ3 brain cortex (laminae III and IV) thalamus striosomes periaqueductal gray rostral ventromedial medulla spinal cord substantia gelatinosa peripheral sensory neurons intestinal tract μ1: analgesia physical dependence μ2:

respiratory depression miosis euphoria reduced GI motility physical dependence μ3:

possible vasodilation

The aim of a study of Ji and Wang from the Cangzhou Central Hospital, was to investigate the role of μ-opioid receptors in acute respiratory distress syndrome and whether their protective effect is mediated via PI3K/AKT/mTOR pathway. What is the main finding and its importance? The findings show that activation of μ-opioid receptors ameliorates lung injury, effects reversed by the PI3K inhibitor, wortmannin.

The main pathology of acute respiratory distress syndrome (ARDS) is the accumulation of inflammatory cells in the lung and increased permeability of vascular endothelial cells. The μ-opioid receptor (MOR) is a G protein coupled receptor, which stimulates angiogenesis and vascular endothelial cell proliferation. In addition, MOR inhibited cell apoptosis via PI3K/Akt signaling pathway. In this study, they aimed to explore the contribution of MOR in ARDS and whether effects are mediated via PI3K/Akt signalling. An ARDS model was established by intra-tracheal instillation of 5 mg k-1 g lipopolysaccharide (LPS). Lung injury was confirmed by hematoxylin and eosin staining, lung wet/dry weight ratio, bronchoalveolar lavage fluid (BALF) protein concentrations, myeloperoxidase (MPO) activity and vascular cell adhesion molecule 1 (VCAM-1) expression. Lung inflammation was determined by assessment of interleukin-1beta (IL-1β) and tumor necrosis factor alpha (TNF-α) concentrations. The protein levels of p-Akt was detected by western blot. Endomorphin-1-activated MORs attenuated LPS-induced lung injury, lung wet/dry weight ratio, BALF protein concentrations, MPO activity, IL-1β and TNF-α levels and VCAM-1 expression, and elevated LPS-induced decreased p-Akt expression. However, the protective effect of MOR activation on lung injury was reversed by the PI3K inhibitor, wortmannin. In conclusion, μ-opioid receptor involvement in LPS-induced ARDS is via the PI3K/Akt pathway [1)].

1)
Ji S, Wang L. The role of μ-opioid receptors in respiratory distress syndrome μ-opioid receptor signalling via PI3K/Akt pathway ameliorates lipopolysaccharide-induced acute respiratory distress syndrome. Exp Physiol. 2019 Jul 4. doi: 10.1113/EP087783. [Epub ahead of print] PubMed PMID: 31272134.