COMMENTARY

Role of Interleukin in Opioid Resistance

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DESCRIPTION

Morphine acts primarily on μ opioid receptors to deliver its potent analgesic effects. For the treatment of acute pain, postoperative pain, and moderate to severe chronic pain, it has been frequently utilised in clinics. However, morphine's clinical use is hampered by a number of unavoidable side effects, including drug dependence and tolerance, respiratory depression, nausea, vomiting, and drowsiness. According to reports, a number of mechanisms, such as desensitisation and internalisation of the opioid receptor, heterodimers of G protein-coupled receptors, activation of the adenosine 3', 5'-monophosphate pathway and the Mitogen-Activated Protein Kinase (MAPK) pathway, among others, contribute to the development of morphine tolerance. However, the available treatments for morphine tolerance management are still insufficient. Cytokines are produced by a wide variety of cells, including immune cells, endothelial cells, and different stromal cells. Considering the nature

In terms of immune response, cytokines can be broadly divided into three groups: Pro-inflammatory signalling, which promotes inflammation, such as Interleukin 1 (IL-1), Interleukin 6 (IL-6), Tumour Necrosis Factor alpha (TNF-α). Anti-inflammatory signalling, which inhibits inflammation, such as Interleukin 4 (IL-4), and common chain (CD131) receptor ligands. Interleukins, tumour necrosis factors, interferons, colony stimulating factors, chemokines, and other cytokines, including TGF-α, could potentially be classified as cytokines. Cytokines have vital roles in inflammation, immunological responses, haematopoiesis, and other important physiologic processes as the powerful and essential mediators in cell signalling. According to reports, the analgesic efficacy of morphine may be impacted by aberrant pro-inflammatory, chemokine, and anti-inflammatory cytokine expressions brought on by long-term morphine treatment.

A group of immune regulatory molecules known as anti-inflammatory cytokines may be to blame for the pro inflammatory cytokine response. Many different cell types, including monocytes, macrophages, activated lymphocytes, and mast cells, release IL-10, an 18-kDa non-glycosylated peptide. IL-10, the first anti-inflammatory cytokine to be identified, is essential for controlling immunological reactions. IL-10 receptor (IL-10R) is a member of the class II cytokine receptor family, just like interferon receptors. The Janus Kinase/Signal Transducer and Activator of Transcription (JAK/STAT) signalling pathway may be triggered when IL-10 selectively binds to IL-10R, producing an anti-inflammatory effect. Previous research has shown that IL-10 can promote the endogenous expression of anti-inflammatory cytokines and decrease the synthesis of pro-inflammatory cytokines including TNF-, IL-1, and IL-6. In addition, IL-10 may promote mastocyte growth and suppress natural killer cell production of IFN. Many cytokines, including IL-4, IL-13, and IFN, could restrict IL-10's expression and be suppressed by the cytokine's own auto-regulation.

IL-10, a powerful immune-regulatory cytokine, has a significant effect on controlling inflammation and the course of the adaptive immune response. In Adjuvant Arthritis (AA) morphine-tolerant rats, which has been demonstrated to be involved in the development of morphine tolerance, is connected to the regulation of serum anti-inflammatory cytokine IL-10. In AA morphine-tolerant rats, the expression of spinal MOR and serum IL-10 both increased. In comparison to the control group, AA rats took significantly longer to develop morphine tolerance. Anti-IL-10 antibody therapy given daily to AA rats may also considerably increase thermal hyperalgesia, reduce spinal MOR expression, and hasten the establishment of morphine tolerance. These findings suggested that spinal MOR expression in AA rats might be modified by the up-regulation of serum IL-10 to prevent the development of morphine tolerance.

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