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**Format:** Abstract**Full text links**Neuroscientist. 2004 Feb;10(1):40-52.

Neuroimmune activation and neuroinflammation in chronic pain and opioid tolerance/hyperalgesia.

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Abstract

One area that has emerged as a promising therapeutic target for the treatment and prevention of chronic pain and opioid tolerance/hyperalgesia is the modulation of the central nervous system (CNS) immunological response that ensues following injury or opioid administration. Broadly defined, central neuroimmune activation involves the activation of cells that interface with the peripheral nervous system and blood. Activation of these cells, as well as parenchymal microglia and astrocytes by injury, opioids, and other stressors, leads to subsequent production of cytokines, cellular adhesion molecules, chemokines, and the expression of surface antigens that enhance a CNS immune cascade. This response can lead to the production of numerous pain mediators that can sensitize and lower the threshold of neuronal firing: the pathologic correlate to central sensitization and chronic pain states. CNS innate immunity and Toll-like receptors, in particular, may be vital players in this orchestrated immune response and may hold the answers to what initiates this complex cascade. The challenge remains in the careful perturbation of injury/opioid-induced neuroimmune activation to down-regulate this process without inhibiting beneficial CNS autoimmunity that subserves neuronal protection following injury.

PMID: 14987447 DOI: [10.1177/1073858403259950](https://doi.org/10.1177/1073858403259950)

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