PubMed

Abstract

Full text links

BenthamScience Full-Text Article

CNS Neurol Disord Drug Targets. 2013 Feb 1;12(1):34-44.

Non-neuronal cell modulation relieves neuropathic pain: efficacy of the endogenous lipid palmitoylethanolamide.

Bettoni I[1], Comelli F, Colombo A, Bonfanti P, Costa B.

Author information

Abstract

We have previously shown that the endogenous lipid palmitoylethanolamide (PEA) induced relief of neuropathic pain through an action upon receptors located on the nociceptive pathway. Recently, it has been proposed that immune cells, in particular mast cells, and microglia, by releasing algogen mediators interact with neurons to alter pain sensitivity thereby contributing to the development and maintenance of chronic pain states. The aim of this work was to explore whether the anti-nociceptive properties of PEA might be accompanied by modulation of these non-neuronal cells. Mice were subjected to a chronic constriction injury model of neuropathic pain and treated with PEA. The data show that at the earlier (3 days) time-point after nerve injury there was a substantial recruitment of mast cells whose activation was not yet pronounced. In contrast, at the later time point (8 days) there was no further increase in mast cell number, but rather a marked activation of these cells. An up-regulation of activated microglia was found in the spinal cord of neuropathic pain mice. PEA delayed mast cell recruitment, protected mast cells against degranulation and abolished the nerve growth factor increase in sciatic nerve concomitantly preserving the nerve from degeneration, while reducing microglia activation in the spinal cord. These findings support the idea that non-neuronal cells may be a valuable pharmacological target to treat neuropathic pain since the current neuronal-direct drugs are still unsatisfactory. In this context PEA could represent an innovative molecule, combining a dual analgesic activity, both on neurons and on nonneuronal cells.

PMID: 23394519 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

LinkOut - more resources

PubMed Commons

PubMed Commons home

0 comments

How to join PubMed Commons