Poster

056. Pain: Inflammatory Mechanisms

Location: Hall A

Time: Saturday, October 19, 2019, 1:00 PM - 5:00 PM

Program #/Poster #: 056.17/J29

Topic: D.03. Somatosensation – Pain

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Title: IB-MECA acute treatment relieves pain in CFA chronic inflammatory model in rats

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Abstract: It is known that IB-MECA compound, an agonist of adenosine A3 receptor (A3AR), is involved with pain relief and modulation in the inflammatory process; however, its action mechanisms are not completely elucidated. The aim of this study was to evaluate the antinociceptive effect of IB-MECA) in a chronic inflammatory pain model, and the involvement of neurotrophins and cytokines central levels in this effect. Chronic inflammatory pain was induced using Complete Freund's Adjuvant (CFA) in the hind paw of male adult Wistar rats. Thermal and mechanical hyperalgesia/allodynia was measured by Hot plate, Von Frey and Randal Selitto tests. Neurochemical measured were brain-derived neurotrophic factor (BDNF), nerve growth factor (NGF), interleukin 1β (IL-1β) and IL-10. The establishment of pain model, decrease of latency withdrawal, was observed 10 and 14 days after CFA injection. And, IB-MECA was effective to revert mechanical and thermal hyperalgesia, in a totally or partially way. We observed CFA pain model effects in IL-1β and IL-10 spinal cord and brainstem levels. Also,we showed that IB-MECA administration in controls increased the interleukin levels. And, we did not find any involvement neurotrophins in this effect, at least those we measured in spinal cord and brainstem of rats. Studies have shown that CFA increases the IL-1β in the hind paw of injection when evaluated acutely after the induction of model. But, there is a lack of knowledge about the neuroinflammatory effects induced by CFA pain model. Also, the adenosine A3

receptor seems to have complexes effects in the central nervous system, with proinflammatory and anti-inflammatory roles, specially in healthy conditions.

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Poster

056. Pain: Inflammatory Mechanisms

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Title: The effect of morphine on post-operative allodynia and toll like receptor 4 mediated inflammation in male and female rats

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Abstract: Our lab has previously shown that a short course of morphine potentiates neuropathic pain in the chronic constriction injury (CCI) model in male and female rats and in a post-operative (i.e. laparotomy) model of pain in male rats. We have further demonstrated that the potentiation of neuropathic pain by morphine is mediated by activation of toll like receptor 4 (TLR4) and downstream activation of the Nod-like receptor protein 3 (NLRP3) inflammasome pathway, which causes release of pro-inflammatory cytokines that mediate pain such as interleukin-1βin male rats. Whether female rats express the same morphine-induced TLR4 activation pathway to the NLRP3 inflammasome in either neuropathic pain or postoperative pain is unknown. In the experiments presented here we have explored the prolonged allodynia following morphine treatment in the post-operative pain model of paw incision, and how this is altered by inhibition of TLR4 by the co-administration of the TLR4 antagonist (+)-naloxone (20mg/kg) along with a 5-day course morphine (5mg/kg) in male and female rats.