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MEETING ABSTRACT Open Access

Functions of NO-GC1 and NO-GC2 in pain processing

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Background

Chronic pain in response to tissue inflammation (inflammatory pain) or nerve injury (neuropathic pain) is often unresponsive to currently available treatments. A large body of evidence indicates that production of nitric oxide (NO) and activation of NO-sensitive guanylyl cyclase (NO-GC) essentially contributes to the processing of chronic pain. NO-GC is a heterodimer consisting of one α subunit (α_1 or α_2) and one β_1 subunit and exists in two isoforms (NO-GC1 and NO-GC2). However, the functional role of NO-GC1 and NO-GC2 in pain processing remains poorly understood. Here, we investigated the expression of NO-GC isoforms in pain-relevant tissues (dorsal root ganglia and the spinal cord) and characterized the nociceptive behavior of mice lacking α_1 or α_2 in models of acute nociceptive, inflammatory and neuropathic pain.

Conclusion

Our behavioral data point to different and partly contrary functions of NO-GC1 and NO-GC2 in the processing of inflammatory and neuropathic pain. The expression of NO-GC isoforms in dorsal root ganglia and the spinal cord is restricted to specific neuronal and non-neuronal cell populations. It remains to be determined which targets mediate the pain-relevant effects of NO-GC1 and NO-GC2.

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