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Spinal cellular and subcellular localization of COX-1 and COX-2 after spinal nerve ligation

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Cyclooxygenase (COX) is involved in the development of neuropathic pain. Recent studies suggested that the development of allodynia associated with neural injury may be partly due to upregulation of COX-1 as well as COX-2. However, the cellular source of COX-1 and COX-2 after nerve injury was not clear. In the present study, we examined the changes of COX-1 and COX-2 proteins expression in spinal cord associated with pain perception, and the cellular sources of COX-1 and COX-2 in the development of allodynia following spinal nerve ligation (SNL). Allodynia was induced by ligation of the left L5 spinal nerves in rats. Postoperative pain-related behavior was quantified by measuring the mechanical paw withdrawal thresholds (PWT) and thermal paw withdrawal latencies (PWL) 7 days following spinal injury. COX-1 and COX-2 immunohistochemistry, with collabeling for cell types, were performed on the spinal cord for cellular source determination. SNL rats displayed significant behavioral thermal and mechanical hypersensitivity ($p < 0.05$). There was an increase in both COX-1 and COX-2 immunoreactivity in the lumbar dorsal horn at 7 days post-surgery on the ipsilateral side of surgery animals compared to the contralateral side. Double immunofluorescence labeling demonstrated that COX-1 immunoreactive cells which co-localized with microglia and neurons were predominantly expressed in nucleus whereas COX-2 was expressed in cytoplasm of neurons only. In conclusion, spinal dorsal horn neurons are the source of COX-2 as well as COX-1, whereas microglia are another important source of COX-1 up-regulation for neuropathic pain after spinal nerve injuries.

Biography

H Q Zhang, after obtaining his PhD from University of New South Wales, has been doing neuroscience research related to traditional Chinese Medicine in School of Chinese Medicine, Hong Kong Baptist University. He has published more than 80 papers.

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