Inducible COX-2 expression is regulated by the ARE-binding proteins in inflammatory response in satellite glial cells.

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Satellite glial cells (SGCs) related to primary sensory neurons are altered structurally and functionally under neuroinflammatory conditions. In neuroinflammation expression of cyclooxygenase-2 (COX-2) in peri-sensory neurons results in the production of prostanoids, which affects sensory neuronal activity and responsiveness and causes hyperalgesia. we have shown the facilitated expression of COX-2 by proinflammatory mediators in cultured dorsal root ganglion (DRG) cells. To evaluate the regulatory system of COX-2 expression in the specific cells we investigated the mechanisms using cultured satellite glial cells (cSGCs).

The cSGCs were cultured by dispersing the isolated rat DRG cells and separated by Percoll density gradient centrifugation. mRNA levels were identified with RT-Real time PCR and protein levels were analyzed with Western blotting.

Synergistic expression of COX-2 by interleukin-1beta and bradykinin was observed in the cSGCs. And then the COX -2 transcriptional activities was just increased in an additive manner by a COX-2 promoter luciferase assay. Thus the post-transcriptional regulations might be involved in the COX-2 mRNA levels. Immunoprecipitated HuR, an RNA-binding protein, in the cSCGs contained more COX-2 mRNA than that of the control.

The aberrant control of COX-2 mRNA turnover in SGCs may be implicated in diseases including chronic neuroinflammation, which results in inflammation-derived hyperalgesia occurred around primary sensory neurons.