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Curcumin, COX-2, and Protein p300/CBP

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LETTERS TO EDITORS

Recently, curcumin has received great interest for its emerging role in pain modulation and management [1]. Its anti-nociceptive mechanism is not clear; however, it is part of numerous mechanisms involved in CX3CR1 expression, Mu and Delta opioid receptors, 5-HT (1A) receptors, TNF-α, etc [1].

Recently, I read a report on curcumin which attenuated the pain behavior and serum COX-2 concentration in a rat model of neuropathic pain [2]. That report was of great interest. I have some additional comments about that study.

First, you explained that the decreased COX-2 level after curcumin treatment is associated with the down regulation of the expression of the NF-κB-regulated gene products such as COX-2 [3]. However, interaction of the RelA subunit of NF-κB with the general co-activator protein p300/CBP is vital for RelA-dependent gene transcription [4]. Moreover, disruption of this interaction deregulates the NF-κB pathway by interfering with its negative feedback loop. Another recent study also showed that treatment with 60 mg/kg of curcumin increased the mechanical threshold, as in your study, and reduced COX-2 gene expression [5]. That study revealed that curcumin treatment downregulated the recruitment and altered the binding of protein p300/CBP at the BDNF and COX-2 promoters. Curcumin seems to alleviate neuropathic pain by inhibiting p300/CBP which acts as a vital co-activator of NF-κB instead of direct down regulation of the expression of NF-κB.

Second, curcumin was used 24 hours before making the CCI model and was continued daily to day 7 post-ligation. However, in a clinical situation, neuropathic pain cannot be easily expected and usually treatment starts after neuropathic pain has developed. Thus, further studies on curcumin as a therapy for neuropathic pain is necessary for application in clinical settings.

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