

Cardamonin attenuated thermal hyperalgesia in rheumatoid arthritis-induced rats: Possible involvement of TNF- α and IL-1 β suppression

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Rheumatoid arthritis (RA) is an autoimmune disease which causes chronic inflammation and pain on the systemic joints. The RA patients often suffer from painful joints especially when flares occur. Pro-inflammatory cytokines are the mediators that play a major role in inducing and maintaining the disease process in RA. Cytokines such as tumour necrosis factor- α (TNF- α) and interleukin-1 β (IL-1 β) are abundantly expressed in RA. They contribute to pain indirectly through generation of inflammatory mediators which act on neurons by binding to highly expressed TNF- α and IL-1 β receptors. The aim of this study was to investigate the inhibitory effect of 2',4'-dihydroxy-6'-methoxychalcone (cardamonin) on RA-induced thermal hyperalgesia and its possible target of pro-inflammatory cytokines. Rat model of rheumatoid arthritis were induced via intra-plantar (i.pl.) complete Freund's adjuvant (CFA, 100 μ l) administration in the right hind paws. RA-induced rats were subjected to Hargreaves plantar test, in which the experimental rats were subjected to infrared heat source to RA-induced hind paw. The withdrawal latency of the paw was recorded as nociceptive response. Six groups of rats (n=6) were given cardamonin (0.625, 1.25, 2.5, and 5.0 mg/kg, i.p.), dexamethasone (3.0mg/kg, i.p.) and vehicle (10ml/kg, i.p.). At the end of the study, rat's serum were collected and enzyme-linked immunosorbent assay (ELISA) was carried to assess the level of systemic TNF- α and IL-1 β . Cardamonin was shown to significantly increase the withdrawal response latencies in the thermal hyperalgesia in the RA rat paws while significantly decrease the systemic TNF- α and IL-1 β in the rats. The present result suggested that cardamonin reduced arthritic pain by suppressing TNF- α and IL-1 β level. This finding showed that cardamonin significantly reduced the nociceptive response in rheumatoid arthritis-induced rats, with possible involvement of TNF- α and IL-1 β suppression.

Keywords: Arthritis, Hyperalgesia, Inflammation, Nociception, in vivo

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