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Anti-nociceptive and anti-inflammatory effects of *Aralia cordata*

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Objectives

Persistent pain states may arise from a number of etiologies, including trauma, disease states, chronic inflammation, amputation, or metabolic disturbances, which alter the processing or transmission of nociceptive and non-nociceptive stimuli in pain pathways. Tissue injury results in inflammatory pain. Various inflammatory mediators are involved in initiating and sustaining pain, and inflammatory cascade. The role of prostaglandins (PG) in mediating nociception and inflammation is well understood and, thus, non-steroidal anti-inflammatory drugs (NSAIDs) are used generally as analgesics and anti-inflammatory agents. PGs are generated during metabolism of arachidonic acid by cyclooxygenase (COX) pathway. The root of *Aralia cordata* Thunb. (AC, Araliaceae) has long been used as a traditional Chinese medicine for rheumatism, lumbago, lameness, indicative of its anti-inflammatory activity. The constituents of this plant were reported, and a scientific approach for these analgesic and anti-inflammatory properties has been performed. Recently, the aerial parts of AC and its components showed potent anti-bacterial and cytotoxic effect and anti-inflammatory activity due to their inhibitory action against COX-1 and COX-2 in *in vitro*. Diterpenes, triterpenes, and saponins were also isolated from the aerial parts of AC. Oleanolic acid (OA), one of the components of AC, was naturally found in various medicinal herbs and traditionally used for anti-inflammatory, analgesic, hepatoprotective and cardiotonic effects. Plant extracts rich in OA have been shown to exhibit anti-nociceptive property. However, there are few reports on the antinociceptive and anti-inflammatory activities of the aerial part of AC in *in vivo*.

Materials and Methods

• Acetic acid-induced writhing test in mice : Mice were administered vehicle or a dose of drug followed 20 min later by an i.p. injection of 0.8% acetic acid. The number of writhes produced in these animals was counted for 30 min.

• Hot-tail flick test in mice : Mice had their tails individually immersed in a water bath maintained at 51 ℃, and the latency to removal was measured.

• Formalin-induced nociceptive responses in mice : Formalin-induced nociception was measured using a method described by Miranda *et al.* (2001) with slight modification.

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• **Formalin-induced paw edema in mice** : Caliper (Vernier caliper 530 series, Mitutoyo, Japan) was used to measure the vertical thickness of the injected paw at the metatarsal level in all subjects.

Results

AC (100 and 200 mg/kg, p.o.) inhibited the acetic acid-induced writhing response but did not protect the thermal nociception in hot-tail flick test in mice. Morphine (5 mg/kg, s.c.), an opioid analgesic, alleviated both the acetic acid-induced writhing response and thermal nociception in hot-tail flick test in mice, while ibuprofen (100 mg/kg, p.o.), a representative NSAID, inhibited only the acetic acid-induced writhing response related to PGE_2 synthesis. In the formalin test, AC (50-200 mg/kg, p.o.) and ibuprofen inhibited phase II response (inflammatory response), but not phase I response (tonic response). Formalin-induced paw edema was evaluated as the index of inflammation. Both AC (100 mg/kg, p.o.) and ibuprofen (200 mg/kg, p.o.) significantly alleviated the formalin-induced paw thickness. In conclusion, it is suggested that the aerial part of AC may be efficacious in the treatment of persistent and/or inflammatory nociceptive states. These results indicate AC peripherally inhibit PG-mediated nociception and inflammation via COX inhibition.

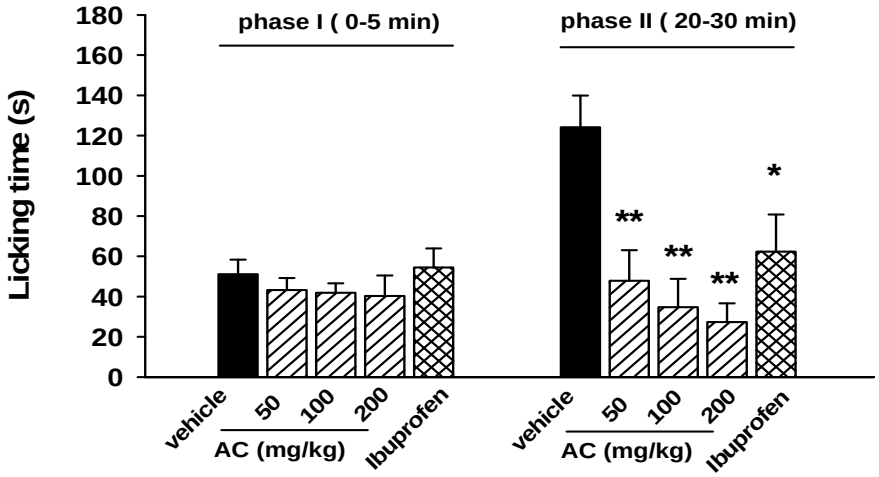

Fig.1. Inhibitory effect of AC on formalin-induced nociceptive response in mice.