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In vitro and *in vivo* anti-inflammatory effects of taheebo, a water extract from the inner bark of *Tabebuia avellanedae*

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Objectives

Tabebuia spp. (Bignoniaceae) are native to tropical rain forests throughout Central and South America and have long been used as a folk medicine to treat bacterial infection, blood coagulation, cancer and inflammatory diseases. In this study, we aimed to demonstrate the ethnopharmacological activity of *T. avellanedae* in various *in vitro* and *in vivo*inflammatory conditions.

Materials and Methods

○ Materials

The dried inner stem bark of *T. avellanedae* was identified by Prof. Walter Radames Accosi. Lipopolysaccharide(LPS, *E. coli* 0111:B4), Arachidonic acid and Croton oil were purchased from Sigma Chemical Co.(St. Louis, MO). An prostaglandin(PG)E_2 EIA kit was purchased from Amersham(Little Chalfont, Buckinghamshire, UK). Fetal bovine serum and RPMI1640 were obtained from GIBCO(Grand Island, NY). RAW264.7 were purchased from ATCC(Rockville, MD).

○ Methods

To do these experiments, anti-inflammatory effects such as PGE2 production, nitric oxide(NO) production, RT-PCR, Immunoblotting and arachidonic or croton oil-induced mouse ear edema were tested according to previous methods using murine macrophage cell lines and male ICR mice (Six-week old).

Results

The results showed that the water extract(taheebo) of *T. avellanedae* significantly suppressed the production of PGE_2 and NO, and blocked the mRNA expression of their catalyzing enzymes (cyclooxygenase[COX)-II] and inducible NO synthase(iNOS), in LPS-stimulated RAW264.7 cells. The blockade of inflammatory mediators by taheebo seemed to be the result of the interruption of ERK activation, according to immunoblotting analysis and the NO assay, where LPS strongly induced the phosphorylation of ERK, and U0126, a selective ERK inhibitor, was found to strongly inhibit PGE_2 production. Similarly, oral administration of taheebo (100 mg/kg) for one week completely diminished mouse ear edema induced by arachidonic acid, an activator of COX-II, but not croton oil, an activator of lipoxygenase. Taken together, these data suggest thatthe ethnopharmacological action of taheebo may be due to its negative modulation of macrophage-mediated inflammatory responses by suppressing PGE_2 production.

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Fig.1 Effect of taheebo on the production of NO and the expression of iNOS in LPS-activated RAW264.7 cells.

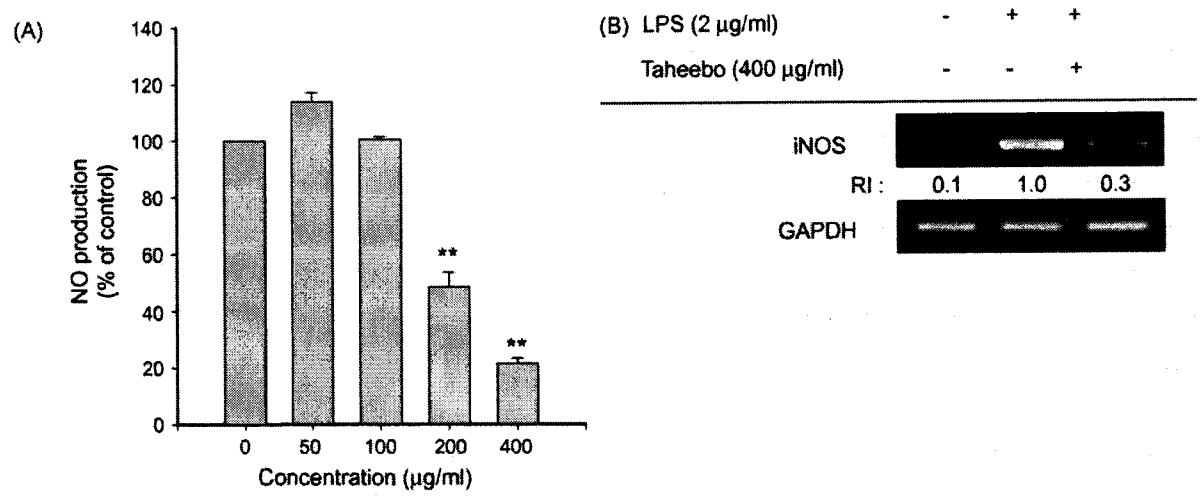

Fig.2 Effect of taheebo on the production of PGE2 and the expression of COX-II in LPS-activated RAW264.7 cells.

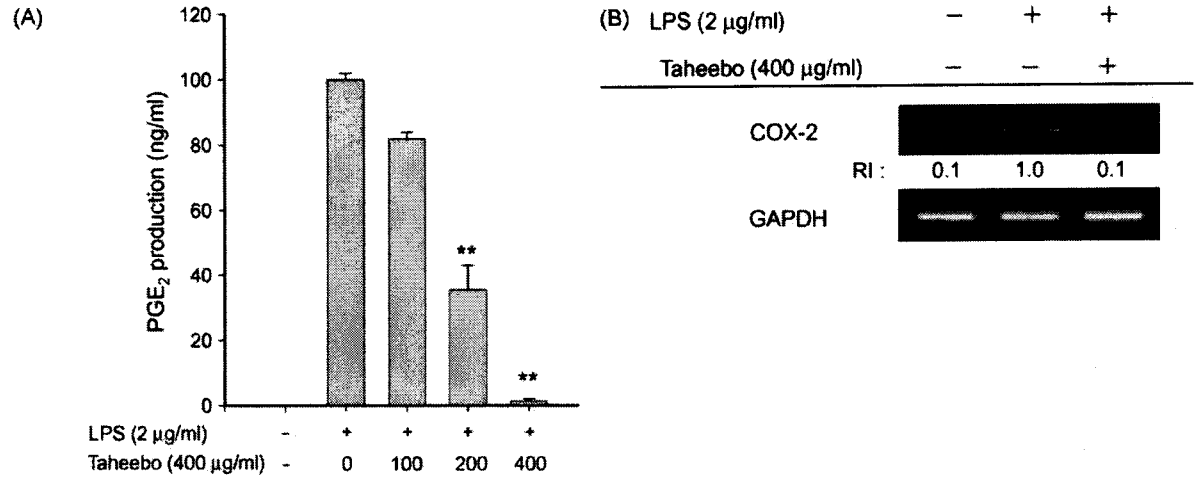

Fig.3 Effect of taheebo on arachidonic acid- and croton oil-treated mouse ear edema.