

POSSES ANTI INFLAMMATORY PROPERTIES

Has anti inflammatory properties to prevent inflammation .

NCBI : Anti-inflammatory activity of an ethanolic *Caesalpinia sappan* extract in human chondrocytes and macrophages

***Caesalpinia sappan* is a common remedy in Traditional Chinese Medicine and possesses diverse biological activities including anti-inflammatory properties.**

Osteoarthritis (OA) is a degenerative joint disease with an inflammatory component that drives the degradation of cartilage extracellular matrix.

In order to provide a scientific basis for the applicability of *Caesalpinia sappan* in arthritic diseases, the present study aimed to assess the effects of an ethanolic *Caesalpinia sappan* extract (CSE) on human chondrocytes and macrophages.

Osteoarthritis (OA) ranks among the major causes of physical disability of elderly patients, thus representing a critical factor in health economics. In contrast to rheumatoid arthritis, OA is conventionally not considered a classical inflammatory arthropathy, but thought to develop from chronic overuse or injury of the joint.

However, evidence has accumulated that, besides mechanical and genetic factors, inflammatory processes within joint tissues contribute to the OA onset and progression. Chondrocytes, the unique cell component of articular cartilage, are embedded in a highly organized extracellular matrix (ECM), comprising collagen type II fibrils and proteoglycans, which confer to the cartilage structural rigidity and protective resiliency.

The present report is first to demonstrate the anti-inflammatory activity of CSE in an in vitro cell model of joint inflammation. CSE can effectively abrogate the IL-1 β -induced over-expression of inflammatory mediators at the transcriptional level in human chondrocytes and macrophages, most likely by inhibiting NF- κ B (p65/p50) signaling.

Blockade of IL-1 β -induced NF- κ B signaling and its downstream pro-inflammatory targets by CSE may be beneficial for reducing cartilage breakdown in arthritis.

It is now accepted that inflammatory processes play a role in the pathophysiology of OA.

Indeed, different studies have shown that joint tissue cells, including synovial fibroblasts, synovial macrophages and chondrocytes, produce proinflammatory cytokines, chemokines and other proinflammatory mediators that will in turn result in an inflammatory environment that drives the upregulation of cartilage-degrading MMPs and ADAMTSs