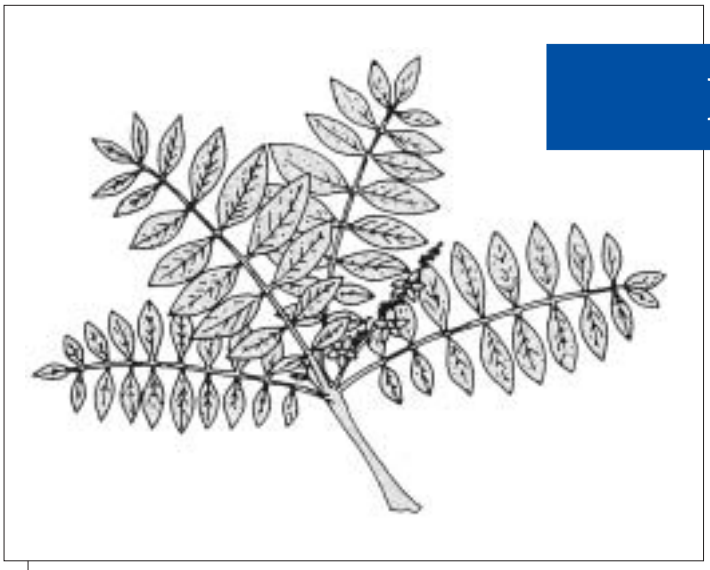

Monograph

Boswellia serrata

Common Name: Frankincense

Description and Constituents

Boswellia serrata is a moderate to large branching tree found in India, Northern Africa, and the Middle East. Strips of bark are peeled away, yielding a gummy oleo-resin which contains oils, terpenoids, and gum. Up to 16 percent of the resin is essential oil, the majority being alpha thujene and p-cymene. Four pentacyclic triterpene acids are also present, with ß-Boswellic acid being the major constituent.

Extracts of this gummy exudate have been traditionally used in the Ayurvedic system of medicine as an anti-arthritic. These gum resins are also known as guggals. S. Nityanand et al showed the guggal of *Commiphora mukul* to be an effective hypolipidemic agent, but it does not have the anti-inflammatory action of the gum resin of *Boswellia serrata*.

Mechanism of Action

Animal studies performed in India showed ingestion of a defatted alcoholic extract of Boswellia decreased polymorphonuclear leukocyte infiltration and migration, decreased primary antibody synthesis,[1,2] and caused almost total inhibition of the classical complement pathway.[3] In an *in vitro* study of the effects of ß-Boswellic acid on the complement system, the extract demonstrated a marked inhibitory effect on both the classical and alternate complement systems.[4] An investigation of Boswellia's analgesic and psychopharmacologic effects noted that it "was found to exhibit marked sedative and analgesic effects" in these animals.[5]

In vitro testing revealed Boswellia specifically, and in a dose-dependent manner, blocks the synthesis of pro-inflammatory 5-lipoxygenase products, including 5-hydroxyeicosatetraenoic acid (5-HETE) and leukotriene B4 (LTB4),[6] which cause bronchoconstriction, chemotaxis, and increased vascular permeability.[7] Other anti-inflammatory plant constituents, such as quercetin, also block this enzyme, but they do so in a more general fashion, as an antioxidant; whereas, Boswellia seems to be a specific inhibitor of 5-lipoxygenase.[8,9] Boswellia has also been observed to inhibit human leukocyte elastase (HLE), which may be involved in the pathogenesis of emphysema. HLE also stimulates mucus secretion and thus may play a role in cystic fibrosis, chronic bronchitis, and acute respiratory distress syndrome.[10,11]

It is known that non-steroidal anti-inflammatory drugs can cause a disruption of glycosaminoglycan synthesis which can accelerate the articular damage in arthritic conditions.[12-15] A recent *in vivo* study examined Boswellia extract and ketoprofen for their effects on glycosaminoglycan metabolism. Boswellia significantly reduced the degradation of glycosaminoglycans compared to controls, whereas ketoprofen caused a reduction in total tissue glycosaminoglycan content.[16]

Clinical Studies

Human clinical studies are woefully lacking for this substance, and need to be conducted to better elucidate its effects in humans, as well as to determine optimal dosing. Animal and *in vitro* studies suggest it is useful for many inflammatory and bronchoconstrictive conditions.

Leukotrienes are suggested to play a role in the inflammatory process of ulcerative colitis. Boswellia extract (350 mg three times daily) was compared to sulfasalazine (1 g three times daily) in ulcerative colitis patients. Patients on the Boswellia extract showed similar improvements as patients on sulfasalazine, although 82 percent of Boswellia patients went into remission, compared with 75 percent on sulfasalazine.[17]

Dosage

For inflammatory or bronchoconstrictive conditions, 400 mg three times per day is suggested.

Toxicity

Toxicity studies of Boswellia in rats and primates showed no pathological changes in hematological, biochemical, or histological parameters at doses of up to 1000 mg/kg. The LD_{50} was established at >2 g/kg.

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