Boswellia serrata pode ser uma boa alternativa para os antiinflamatórios não hormonais

Boswellia serrata: an overall assessment of in vitro, preclinical, pharmacokinetic and clinical data.

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Abstract

Non-steroidal anti-inflammatory drug (NSAID) intake is associated with high prevalence of gastrointestinal or cardiovascular adverse effects. All efforts to develop NSAIDs that spare the gastrointestinal tract and the cardiovasculature are still far from achieving a breakthrough. In the last two decades, preparations of the gum resin of Boswellia serrata (a traditional ayurvedic medicine) and of other Boswellia species have experienced increasing popularity in Western countries. Animal studies and pilot clinical trials support the potential of B. serrata gum resin extract (BSE) for the treatment of a variety of inflammatory diseases like inflammatory bowel disease, rheumatoid arthritis, osteoarthritis and asthma. Moreover, in 2002 the European Medicines Agency classified BSE as an 'orphan drug' for the treatment of peritumoral brain oedema. Compared to NSAIDs, it is expected that the administration of BSE is associated with better tolerability, which needs to be confirmed in further clinical trials. Until recently, the pharmacological effects of BSE were mainly attributed to suppression of leukotriene formation via inhibition of 5-lipoxygenase (5-LO) by two boswellic acids, 11-keto-β-boswellic acid (KBA) and acetyl-11-keto-β-boswellic acid (AKBA). These two boswellic acids have also been chosen in the monograph of Indian frankincense in European Pharmacopoiea 6.0 as markers to ensure the quality of the air-dried gum resin exudate of B. serrata. Furthermore, several dietary supplements advertise the enriched content of KBA and AKBA. However, boswellic acids failed to inhibit leukotriene formation in human whole blood, and pharmacokinetic data revealed very low concentrations of AKBA and KBA in plasma, being far below the effective concentrations for bioactivity in vitro. Moreover, permeability studies suggest poor absorption of AKBA following oral administration. In view of these results, the previously assumed mode of action - that is, 5-LO inhibition - is questionable. On the other hand, 100-fold higher plasma concentrations have been determined for β-boswellic acid, which inhibits microsomal prostaglandin E synthase-1 and the serine protease cathepsin G. Thus, these two enzymes might be reasonable molecular targets related to the anti-inflammatory properties of BSE. In view of the results of clinical trials and the experimental data from in vitro studies of BSE, and the available pharmacokinetic and metabolic data on boswellic acids, this review presents different perspectives and gives a differentiated insight into the possible mechanisms of action of BSE in humans. It underlines BSE as a promising alternative to NSAIDs, which warrants investigation in further pharmacological studies and clinical trials.

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