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INFLAMMATION/IMMUNITY/MEDIATORS

Effects of *Boswellia serrata* in mouse models of chemically induced colitis

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Extracts from *Boswellia serrata* have been reported to have anti-inflammatory activity, primarily via boswellic acid-mediated inhibition of leukotriene synthesis. In three small clinical trials, boswellia was shown to improve symptoms of ulcerative colitis and Crohn's disease, and because of its alleged safety, boswellia was considered superior over mesalazine in terms of a benefit-risk evaluation. The goal of this study was to evaluate the effectiveness of boswellia extracts in controlled settings of dextran sulfate- or trinitrobenzene sulfonic acid-induced colitis in mice. Our results suggest that boswellia is ineffective in ameliorating colitis in these models. Moreover, individual boswellic acids were demonstrated to increase the basal and IL-1 β -stimulated NF- κ B activity in intestinal epithelial cells in vitro as well as reverse proliferative effects of IL-1 β . We also observed hepatotoxic effect of boswellia with pronounced hepatomegaly and steatosis. Hepatotoxicity and increased lipid accumulation in response to boswellia were further confirmed in vitro in HepG2 cells with fluorescent Nile red binding/resazurin reduction assay and by confocal microscopy. Microarray analyses of hepatic gene expression demonstrated dysregulation of a number of genes, including a large group of lipid metabolism-related genes, and detoxifying enzymes, a response consistent with that to hepatotoxic xenobiotics. In summary, boswellia does not ameliorate symptoms of colitis in chemically induced murine models and, in higher doses, may become hepatotoxic. Potential implications of prolonged and uncontrolled intake of boswellia as an herbal supplement in inflammatory bowel disease and other inflammatory conditions should be considered in future clinical trials with this botanical.

dextran sulfate; trinitrobenzene sulfonic acid; liver; steatosis; microarray

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