Effects of ethyl acetate of *Baccharis dracunculifolia* DC in TNBS-induced colitis in rats.

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**Introduction:** Inflammatory bowel disease (IBD) is a chronic inflammatory condition of the gastrointestinal tract divided into two major categories: ulcerative colitis (UC) and Crohn's disease (CD). It’s an etiology remains unknown and medical therapy used for the treatment can induce remission, but in the long-term it may result in adverse side effects and not represent the cure of the disease. Therefore, the development of new drug treatments is an important goal to IBD.

*Baccharis dracunculifolia* (Asteracea), is the main botanical source of Brazilian green propolis, a natural product used to improve health. The ethyl acetate extract of this plant is rich in caffeic acid, p-coumaric acid, aromadendrin-4-O-methyl ether, 3-prenyl-p-coumaric acid, 3,5-diprenyl-p-coumaric acid and baccharin. This extract was effective scavenger of DPPH with the EC50 value of 63.53 vs. 1.89 µg/ml of galic acid. Previous study in our research group showed that this extract presented improvement in experimental model of inflammatory bowel disease induced by trinitrobenzenesulfonic acid (TNBS) in rats. The aim of this study was to investigate by which means this extract acts.

**Material and Methods:** Wistar rats with two mouths of age were pre-treated orally for 5 days with 5 or 50 mg/Kg by *B. dracunculifolia* extract or azathioprine (2mg/Kg), the reference drug, before the colitis induction rectally by 10mg of TNBS in 50% etanol. (Macroscopic (score, extension of lesion, colonic weight/length) and biochemical parameters (gluthathione, myeloperoxidase, alkaline phosphatase, TNF-α and INF-γ) were evaluated.

**Results/Discussion:** The doses of 5 and 50 mg/Kg reduced the colon weight/length and the myeloperoxidase activity. Additionally, the dose of 50 mg/Kg reduced colonic TNF-α levels. The reference drug only reduced the myeloperoxidase activity.

**Conclusion:** The primary mechanism of *B. dracunculifolia* anti-inflammatory actions appears to be by inhibition of myeloperoxidase activity and immunomodulation via suppression of TNF-α production.

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