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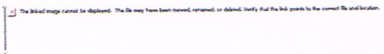

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Analysis of chemical composition and its analgesic and anti-inflammatory activity of essential oil of sintoc bark (*Cinnamomum sintoc* bl.) using in vivo methods

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ABSTRACT

Sintoc bark (*Cinnamomum sintoc* Bl) belongs to Lauraceae (the laurel family). It has been used empirically for a treatment for swelling caused by insects' bites. In this study, the research examined the analgesic activity and anti-inflammation of essential oil of sintocbark using in vivo methods. The mechanism of anti-inflammation was predicted using molecular docking against COX-2. Essential oil of *sintoc* bark was collected by distilling through steam distillation, and then analyzed by GC-MS. Analgesic and anti-inflammatory activity was examined by *in vivo*, which were conducted by writhing and carrageenan-induced methods, respectively. The findings showed that the tested sintoc bark oils contained 36 components of essential oil with eugenol (38.38 %) as a major compound. In the *in vivo* experiments, sintoc bark oils with doses 0.005 mL, 0.010 mL, and 0.020 mL/20g body weight significantly ($p<0.05$) reduced the number of writhing of mice when compared to negative control group. All of doses of sintoc bark oils gave significantly affect (confidence level 99 %) compare to negative control. Sintoc oil with dose 0.2 ml/200g had the strongest inhibition compare to positive control (indometasin 10 mg/kg body weight). The molecular docking results indicated that the compounds of aryl propanoid were generally potential to inhibit COX-2.

INTRODUCTION

Inflammation is a mechanism of the body's defense caused by the tissue response to such detrimental effects-both local or inside the body (Nathan, 2002). The effects can be devastating to physics, chemistry, bacteria, parasites and so forth. Physical damage can be caused by high temperature, light and radiation, including foreign objects inside the organ, or any causes that prompt devastating effects. Strong acids, strong bases and toxins immerse in chemical causes. Pathogenic bacteria of *Streptococcus, Staphylococcus* and *Pneumococcus* genus, for instance, are also devastating (Khansari*et al.*, 2009). Inflammatory reaction can be observed from clinical symptoms around affected tissue, such as increase in heat (calor), reddish spots indication (rubor), pains (dolor) and swelling (tumor) along with itching. It is followed by the changes in structural tissues that lead to loss of functions. Those damaged cells release inflammatory mediators, such as, histamine, bradykinin, serotonin, prostaglandins, and leukotrienes. The process of inflammation causes a change in blood flow, an increased permeability of blood vessels, or a damage of tissue through an activation and migration of leukocytes by synthesizing reactive oxygen derivatives and synthesis of inflammatory mediators locally. Oxidative stress caused by free radicals potentially have physiological or biochemical effects in metabolic disorders that lead to the death of cells. It suggests that antioxidants play a role to heal an inflammation. A synthesis of inflammatory mediator is inducted by phospholipase, cyclooxygenase (COX) and lipooksigenase (LOX) enzymes (Gilroy *et al.*, 1999). Arachidonic acid in cell membranes will be esterified into phospholipids, while the others are in the form of complex lipids. In prostaglandins biosynthesis, arachidonic acid will be released from lipid storage cells byacyl hydrolase.

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