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Inhibitory Activity of *Kaempferia galanga* and *Hibiscus sabdariffa* on the Rate of PGH_2 Formation

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ABSTRACT
Cyclooxygenase (COX) or Prostaglandin H_2 Synthase (PGHS) is the enzyme that catalyzes the first two steps in the biosynthesis of prostaglandins from the substrate, arachidonic acid. The NSAIDs work by inhibiting both COX isoforms, thus the conversion of arachidonic acid into prostaglandin is disturbed. In our country, kaempferia rhizome (*Kaempferia galanga*) and roselle calyx (*Hibiscus sabdariffa*) are used to reduce inflammation by consuming herb tea of these plants. This work studied the inhibitory activity of kaempferia rhizome and roselle calyx on the rate of prostaglandin formation by measuring the absorbance of TMPD (tetramethyl-p-phenyldiamine) oxidized by the extracts, against time. The plants were collected from Manoko plantation in West Java. Phytochemical screening showed that the rhizome contains quinones and terpenes, while the calyx contains polyphenol, flavonoid, quinone and saponin. 100 g of dried rhizome and calyx were separately boiled in 1 L of distilled water for 15 min at 90°C, freeze-dried and dissolved in ethanol 96%. Both *Kaempferia galanga* and *Hibiscus sabdariffa* showed inhibition on PGH_2 formations. The rate of PGH_2 formations on COX-1 was lower than on COX-2. These plants could be further developed for anti-inflammatory drug.

Key words: Anti-inflammatory agents, cyclooxygenase, *Hibiscus sabdariffa*, *Kaempferia galanga*, PGH_2, prostaglandin

INTRODUCTION

Cyclooxygenase (COX) or Prostaglandin H_2 Synthase (PGHS) is the enzyme that catalyzes the first two steps in the biosynthesis of prostaglandins from the substrate, arachidonic acid. These are the oxidation of arachidonic acid to the hydroperoxy endoperoxide, PGG_2 and its subsequent reduction to the hydroxy endoperoxide, PGH_2. The PGH_2 is transformed by a range of enzymes and nonenzymatic mechanisms into the primary prostanoids, PGE_2, $PGF_{2\alpha}$, PGD_2, PGI_2 and TXA_2 (Vane *et al*., 1998). The main reason for classifying COX-1 and COX-2 as physiological and pathological, respectively, is that COX-2 is only expressed when it is induced by stimuli and therefore, it is associated with inflammation.

NSAIDs work by inhibiting both COX isoforms, thus the conversion of arachidonic acid into prostaglandin is disturbed (Katzung, 2007). All NSAIDs in clinical use have been shown to inhibit COX, leading to a marked reduction in PG synthesis. The inhibition by aspirin is due to irreversible acetylation of the cyclooxygenase component of COX. In contrast, NSAIDs like indomethacin or ibuprofen inhibit COX reversibly by competing with the substrate, arachidonic acid, for the active site of the enzyme (Vane *et al*., 1990).

Selective inhibition of COX-2 promises to provide NSAIDs with increased safety and has already become a purposeful approach. A publication by Stubanus and colleague provides evidence suggesting that COX-2 inhibitors impair renal function and cause sodium retention in patients with mild pre-existing renal failure and presumably also in some elderly patients with volume depletion (Stubanus *et al*., 2000).

According to Nomura *et al*. (2003) and Jachak *et al*. (2010), plants with secondary metabolites classified as