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The pathophysiological roles of COX-1 and COX-2 in the intestinal smooth muscle contractility under the anaphylactic condition

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ABSTRACT

Various inflammatory mediators released from antigen-activated mast cells are considered to play a key role in the pathogenesis of food allergy. The aim of the present study was to determine the mechanisms underlying the antigen-induced anaphylactic responses in the rat colons. Wistar rats were sensitized by intraperitoneal injection of ovalbumin (OVA). The contractilities of isolated proximal colons of the sensitized rats were studied in the organ bath. OVA challenges of sensitized tissues induced prolonged contractile responses. The antigen-induced contractions were greatly reduced by mast cell stabilizer doxantrazole (10 μM). However, the contractions were resistant to histamine H1 receptor antagonist and prostaglandin D_2 receptor antagonist. In contrast, non-selective cyclooxygenase (COX) inhibitor indomethacin (1 μM) significantly reduced the contractions by 61.0%. Furthermore, selective COX-1 inhibitor FR122047 (10 μM) as well as selective COX-2 inhibitor NS-398 (10 μM) significantly inhibited the contractions by 50.1% and 50.3%, respectively. Nevertheless, the transcript levels of COX-2 as well as COX-1 were not upregulated by OVA in the proximal colons of the sensitized rats. The present results indicate that de novo arachidonic acid metabolites synthesis by constitutive COX-1 as well as constitutive COX-2 within mast cells contribute to the altered smooth muscle contractilities in the colons under the anaphylactic condition.

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