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**Red Ginseng modulates *Helicobactor pylori* -induced 5S-HETE production in Human Gastric Epithelial Cells**

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Arachidonic acid metabolites like prostaglandins (PGs) and hydroxyeicosatetraenoic acids (HETEs) have been considered as pivotal mediators in *Helicobacter pylori (H.pylori)*-induced inflammatory responses, which are mainly metabolized by two distinct enzymes, cyclooxygenase (COX) and lipoxygenase (LOX). While COX has become known to play a central role in gastrointestinal inflammation and carcinogenesis, LOX is still unclear. In the present study, we evaluated the roles of LOX and its metabolites in *H. pylori*-induced host responses and further a potential preventive effect of red ginseng extract (RGE) on the gastropathy. We observed that *H.pylori* stimulated translocation of $cPLA_2$ from cytoplasm to nucleus and the biosynthesis of HETEs as a predominant form of 5S-HETE in gastric epithelium among the LOX metabolites. Notably, RGE pretreatment as well as the LOX inhibitors, NDGA and Geraniin, had a strong suppression activity of *H. pylori*-induced 5-LOX production and signaling. Moreover, the deterrent action of the inhibitors was related with down-expression of pro-inflammatory mediators such as IL-8 and TNF-α in both *H. pylori*-infected gastric epithelial cells and macrophage cells. Taken together, RGE inhibited preferential metabolize of arachidonate 5S-HETE via 5-LOX inhibition in *H.pylori*-infection, which may contribute to the protection of *H.pylori*-related gastropathy.

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