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Akinori Yanaka 1

Sulforaphane enhances protection and repair of gastric mucosa against oxidative stress in vitro, and demonstrates anti-inflammatory effects on Helicobacter pylori-infected gastric mucosae in mice and human subjects

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Abstract

Helicobacter pylori infection induces oxidative stress on gastric mucosa, thereby causing mucosal damage, retarding mucosal repair, and eventually inducing gastric cancer. Cells can survive against chronic oxidative stress by enhancing activities of antioxidant enzymes, thereby protecting cells from DNA damage. Recent studies have clearly shown that the genes encoding nrf2 (NF-E2 p45-related factor-2) and keap1 (Kelch-like ECH-associated protein 1) play an important role in the induction of antioxidant enzymes against oxidative stress. In this paper, we will first describe the cellular mechanisms by which the nrf2-keap1 system contributes to induction of a variety of antioxidant enzymes during exposure to oxidative stress. Secondly, we will also mention beneficial effects of a natural compound sulforaphane, an isothiocyanate family, rich in broccoli sprouts, on gastric mucosa. Sulforaphane stimulates nrf2 gene-dependent antioxidant enzyme activities, thereby protecting cells from oxidative injury. Finally, we will show our data on the effect of sulforaphane, a natural chemical compound rich in broccoli sprouts, on protection and repair of gastric mucosa against oxidative stress, and anti-inflammatory effects on gastric mucosa during H. pylori infection, which appears to be closely related to chemoprotection against gastric cancer induced by H. pylori-infection.

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