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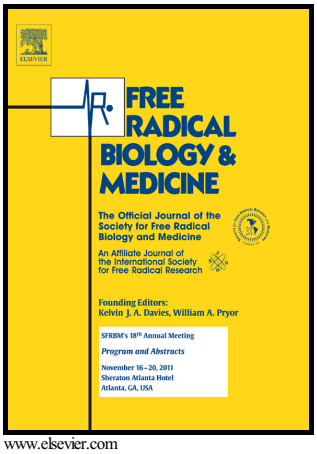

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REDOX SIGNALING IN THE GASTROINTESTINAL TRACT

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ABSTRACT

Redox signaling regulates physiological self-renewal, proliferation, migration and differentiation in gastrointestinal epithelium by modulating Wnt/β-catenin and Notch signaling pathways mainly through NADPH oxidases (NOXs). In the intestine, intracellular and extracellular thiol redox status modulates the proliferative potential of epithelial cells. Furthermore, commensal bacteria contribute to intestine epithelial homeostasis through NOX1- and dual oxidase 2-derived reactive oxygen species (ROS). The loss of redox homeostasis is involved in the pathogenesis and development of a wide diversity of gastrointestinal disorders, such as Barrett's esophagus, esophageal adenocarcinoma, peptic ulcer, gastric cancer, ischemic intestinal injury, celiac disease, inflammatory bowel disease and colorectal cancer. The overproduction of superoxide anion together with inactivation of superoxide dismutase are involved in the pathogenesis of Barrett's esophagus and its transformation to adenocarcinoma. In *Helicobacter pylori*-induced peptic ulcer, oxidative stress derived from the leukocyte