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ABOUT THE COVER

Chronic inflammation induces histopathologic progression of the stomach epithelium leading to the development of metaplasia followed by gastric adenocarcinoma. Inflammation of the gastric epithelium, which produces high levels of reactive oxygen species (ROS), results in a gradual loss of parietal cells and their replacement with proliferative metaplastic cells, suggesting that the inflammation-associated ROS plays a role in the disruption of homeostasis of the gastric epithelium. However, the role of ROS and its downstream signaling in gastric carcinogenesis has remained unknown. The cover illustration depicts the phosphorylated (activated) form of p38MAPK (green) as well as parietal cells (H^+ dik-ATPase, red) in normal stomach tissue exposed to the hydrogen peroxide in vitro (nuclei are counterstained in blue). As shown in the yellow signal (red and green overlay), the oxidative stress–dependent activation of p38MAPK is triggered selectively in parietal cells. For more information on the potential mechanisms underlying the oxidative stress–dependent parietal cells loss and consequent gastric carcinogenesis, see the article by Seishima et al. (beginning on page 492).
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