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Helicobacter pylori and Stress Augment the Gastric Mucosal Lesion ?

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We hypothesized that if *H. pylori* infection or stress can induce oxidative damage in the stomach, the antioxidant can modulate the pathogenic course. We infected the SD rats or Mongolian gerbils with SS1 strain of *H. pylori* (VacA+, CagA+) and impose the water immersion restraint stress (WIRS) on experimental animals. WIRS was imposed for 30, 120 and 480 min, respectively in each group and sacrificed animals for gross and microscopic lesion scores, measurement of MDA, iNOS, GSH, transcription factors (NF-κB and AP-1), and RPA (cytokines and chemokines) from homogenated gastric mucosa, respectively. Significantly elevated levels of MDA, iNOS and decreased levels of GSH were observed in WIRS and *H. pylori* infected group compared to WIRS or *H. pylori* infection alone group. All translated and transcriptional levels of cytokines and chemokines (IL-8, MIF, RANTES) were increased significantly in animals imposed with both WIRS and *H. pylori* infection. Based on these results, DA-9601, a new antiulcerant possessing antioxidative and cytoprotective actions, was additionally investigated the effect of antioxidants. Significant attenuation of gross and microscopic lesion scores was noted in animals suffered from WIRS and *H. pylori* infection treated with antioxidant compared to non-treated group and even stress alone group. Conclusively, oxidative stress was the fundamental basis of augmentation of gastric lesions by both stress and *H. pylori* infection and antioxidant should be considered in the treatment of *H. pylori*-associated gastric lesion under stressful condition.