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GENE-SPECIFIC OXIDATIVE DNA DAMAGE IN HELICOBACTER PYLORI INFECTED HUMAN GASTRIC MUCOSA

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Abstract To study the status of oxidative DNA damage in Helicobacter pylori infection in more details, gene-specific oxidative DNA damage was investigated by examining oxidative DNA damage to individual genes. This was done by determining the loss of PCR product of a targeted gene before and after gastric mucosal DNA was treated with 8-hydroxyguanine glycosylase, which cleaves DNA at the 8-hydroxyguanine residues. The results showed that, of the 5 genes tested, the genes of P53, insulin-like growth factor II receptor and transforming growth factor-beta receptor type II showed significant oxidative DNA damage in H. pylori-positive tissues, and that the BAX and beta-ACTIN genes relatively undamaged. These results suggest that in the case of H. pylori infection, oxidative DNA damage does not occur homogeneously throughout the genomic DNA, but rather occurs in a gene-specific manner. We conclude that the progressive accumulation of preferential oxidative DNA damage in certain genes, such as P53, is likely to contribute to gastric carcinogenesis.

keyword : Helicobacter pylori, 8-hydroxydeoxyguanosine, Gene-specific DNA damage, P53, gastric carcinogenesis