

A Known Gene in a Novel Location May Be Related to Gastric Carcinogenesis

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Abstract

Sir - The role of genomic changes in the pathogenic *Helicobacter pylori* (HP) in inducing benign structural damage and neoplasia to the gastric epithelia has been well described (1). The genomic changes in the gastric epithelia induced by the pathogenetic HP has also been discovered (2). Malignant derangement of cells and tissues are the result of uncontrollable clonal proliferation which in turn is due to the genomic changes occurring at the molecular level. Thus, it is understandable to appreciate the genomic changes that take place in the transformation of normal gastric epithelia into malignancy induced by HP.

Most gastric epithelial malignancy which is related to *Helicobacter pylori* infection is preceded by chronic atrophic gastritis (3). This is one example of where the chronic inflammatory process of the tissues harboring epithelia giving rise to the epithelial malignancy. These transformational versus neoplastic are ample evidence of the fuzzy dichotomy of inflammatory versus neoplastic processes. The gross phenotypic manifestations of inflammatory and neoplastic cells and tissues may be easily distinguishable. However, the subcellular processes especially at the molecular levels may have significant overlapping between inflammation and neoplasm. Genomic changes in the gastric epithelia that may be instrumental in the process of malignant transformation could well be happening during the inflammatory phase. These genetic alterations constitute host manifestations induced by *Helicobacter pylori* infection (2). The detection of these genetic expressions are made possible by various molecular techniques such as In-situ hybridization, DDRT-PCR analysis and DNA microarrays enhanced further by the utilization of the ever expanding bioinformatics technology (4).

Recently, using the DDRT-PCR analysis technique and with reference against the genome database (Genbank NIH, USA), we have discovered an overexpression of ubiquinol-cytochrome C reductase complex gene which is located on Chromosome 22 (Chrom 22cen-q12.3) in a specimen of inflamed antral (gastric) tissue from a patient who was suffering from chronic epigastric pain but *Helicobacter pylori* was negative on histopathological examination. To our knowledge, this is perhaps the first report in the English literature of this expressed gene discovered in a "novel" location in the absence of *Helicobacter pylori* infection. Further cases and specimens need to be studied. It is interesting to study further whether this mitochondrial metabolic gene expression is significant in the process of transformation from inflammation to malignancy in gastric epithelial tissues. It remains to be elucidated whether this phenomenon is essentially genetic or epigenetic in nature.

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