Short description of molecular pathogenesis of gastric cancer.

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Introduction

Gastric Cancer (GC) is the fifth most common cancer and the third most common cause of cancer- related death worldwide. Incidence and mortality for GC are mostly seen in South America, East Asia, and Eastern Europe. *Helicobacter pylori* infection, smoked and fried food, diets low in fruit and vegetables, high salt intake, geographical location, genetic factors, age, and sex are considered risk factors for the development of stomach cancer. Stomach cancer is twice as likely to occur in men than in females. Eventhogh, a decline in the incidence and mortality rates of this cancer is observed, more gastric cancer cases is expected in the future due to ageing populations. Moreover, improved socio-economic conditions have contributed to the reduction in the prevalence of *Helicobacter pylori*, the major cause of gastric cancer. On the other hand, the fact that young people with high socio-economic status are diagnosed with gastric cancer in developed countries indicates the ongoing transitions in the epidemiology of gastric cancer.

Approximately, 10% of patients with gastric cancer show familial aggregation and 1–3% of them have germline mutations. Hereditary diffuse type gastric cancer, familial intestinal gastric cancer, and gastric adenocarcinoma with proximal polyposis of the stomach are the hereditary forms of gastric cancer. In addition to histological classification of gastric cancer genome-wide analyses of DNA copy number alterations, mutations, mRNA, miRNA, and protein patterns proposes four molecularly distinct gastric cancer subtypes categorised as Epstein-Barr virus-positive (EBV+), Micro Satellite Instable (MSI), genomically stable, and chromosomal unstable (CIN) forms. Currently, MSI and EBV+ gastric cancer subtypes is of the clinical importance. Gain or loss of whole chromosomes (aneuploidy) or parts of chromosomes Loss Of Heterozygosity (LOH), and translocations are the signitures of CIN in sporadic gastric tumors. In 15-20% of gastric cancers, with a higher frequency in familial cases and amplifications, DNA replication errors result in MSI. Mutational activation and/or amplification of oncogenes, mutations, LOH and epigenetic inactivation of tumor supressor genes, mutational inactivation and downregulation of genes encoding cell-adhesion molecules, inactivation of cell cycle regulators and aberrant expression of growth factors and cytokines play a pivotal role in the pathogenesis of gastric cancer. Gastric cancer due to *Helicobacter pylori* infection is associated with bacterial virulence, genetic polymorphism of hosts, and environmental factors. Cytotoxin-Associated Gene A (CagA) pathogenicity island that is encoding a 120–140 kDa CagA protein, which is an oncoprotein of most *Helicobacter pylori* strains. This protein is phosphorylated by SRC family kinases. Phosphorylated CagA activates growth factor receptors, increases proliferation, promotes invasion and angiogenesis, and inhibits apoptosis. It is well understood that, the gastric epithelium is continuously exposed to Reactive Oxygen Species (ROS) within the gastric lumen as a consequence to ingested food, smoke, and inflammation due to *Helicobacter pylori* infection. The dynamic balance between cell proliferation and apoptosis is critical for maintaining mucosal homeostasis. Decreased apoptosis as well as increased proliferation may favor the carcinogenic process. Prolonged survival of abnormal cells can support the accumulation of sequential genetic mutations, changes in gene expression profiles and protein structure and function, which can result in gastric tumor promotion.

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