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Perspective

Pathophysiology and Risk Factors involved in Gastro Intestinal Cancer

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ABOUT THE STUDY

According to statistics from 2020, there were 1,806,590 new cases of cancer and 606,520 cancer deaths in the country, with gastrointestinal cancer being one of the leading causes of death. Colorectal cancer is the most prevalent and deadly kind of cancer. The second is pancreatic cancer, which has a 9% five-year survival rate in America. These followed by malignancies of the stomach, oesophagus, and liver. A High-Fat Diet (HFD) is the primary cause of obesity, which raises the risk of gastrointestinal cancer. It has high levels of fatty acids but has little fiber, vitamin, or mineral content. With the accelerated pace of life and economic growth in recent years, obesity has emerged as a major global health issue, increasing the risk of a number of chronic illnesses. The rate of obesity worldwide is predicted to increase to 18% for men and 21% for women by 2025. The global burden of gastrointestinal cancer may rise as obesity becomes more common.

Gut bacteria has achieved a new peak in recent years with the discovery of the metagenome and macro transcriptome. These organisms are dynamic and subject to influences from medications, food, lifestyle, genetics, and the environment. Researchers have shown that gut microbes affect extra-intestinal conditions like hepatic illness and pancreatic disease in addition to intestinal diseases like inflammatory bowel disease and CRC. Additionally, studies have shown that HFD can drastically change the gut microorganisms. Intestinal microbes may be crucial in the aetiology of gastrointestinal cancers brought on by HFD. The relationship between HFD, gut bacteria, and gastrointestinal malignancies as well as possible mechanisms by which HFD alters the characteristics of intestinal microbes to promote the growth of gastrointestinal tumours.

Esophagus cancer

Esophageal cancer incidence was discovered to be greater in rats fed HFD in 1994, pointing to a possible link between HFD and the disease. By altering the mice's bile acids, particularly taurocholic acid and tauroursodeoxycholic acid, HFD can enhance the likelihood that mice will develop Barrett's oesophagus and esophageal cancer.

Gastric cancer

There have been numerous epidemiological studies that suggest eating fat may increase the risk of stomach cancer. Due to its role in angiogenesis, apoptosis, cell proliferation, and migration, leptin is expected to play a significant impact in obesity-related gastrointestinal cancers. Additionally, *via* controlling mTOR, STAT3 and ERK-dependent pathways, PI3K-dependent pathways, and MAPK-dependent pathways, it has been demonstrated to encourage the synthesis of mucin and the development of gastrointestinal tumours. By inhibiting suppressors of cytokine signaling 3 in gastrointestinal epithelial cells, increasing the expression of ectopic molecules related to intestinal epithelium, such as intestinal mucin 2 and Paneth cell marker PLA2, and decreasing the expression of the transcription factor, excessive leptin and leptin signaling activation can cause gastric tumours.

According to a study, stomach wall cells suffered mitochondrial damage and an increase in mucosal thickness throughout the course of 8-20 weeks of HFD eating. HFD may raise the degree of O-Glc-N-acylation, which aided in the transcriptional activation of CD36, and could offer enough energy for metastasis. Upregulation of CD36 causes gastric cancer cells to take in more fat, creating a vicious cycle that encourages metastasis.

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