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Opinion Article

Molecular Basis of Alcohol-Related Gastric Cancer

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DESCRIPTION

For a number of gastrointestinal malignancies, alcohol usage is a substantial and potentially modifiable risk factor. It is often undervalued how much alcohol contributes to the burden of gastrointestinal cancer. One method to lessen the incidence of gastrointestinal cancer is to address high-risk alcohol use. Hepatocellular carcinoma, breast cancer, Colorectal Cancer (CRC), oesophageal (squamous cell) and gastric cancer, as well as nasopharynx and most likely, pancreatic cancer are all directly linked to alcohol consumption. The number of malignancies linked to alcohol consumption is continually expanding. Even occasional drinking can increase the chance of developing cancer, especially when combined with tobacco use or being overweight. Recently, it was determined that high-income countries should consume no more than 100 g of alcohol per week to have the lowest risk of all-cause mortality. Other than myocardial infarction, there are no risk thresholds at which lower alcohol consumption ceases to be linked to a lower risk of disease.

Alcohol-mediated cancer development

A group 1 human carcinogen, according to the International Agency for Research on Cancer (IARC), is acetaldehyde generated from alcoholic beverages. This IARC conclusion, which is based on gene-epidemiologic data on alcohol consumers with ALDH2 deficiencies, is particularly concerned about alcohol-related upper digestive tract malignancies. According to the WHO, alcohol usage results in more than 3 million fatalities annually (5.3% of all deaths globally), with cancer accounting for around 13% of those deaths. Alcoholic beverages mostly include ethanol. Alcohol (ethanol) is degraded by Alcohol Dehydrogenase (ADH) into acetaldehyde after ingestion, which is then converted to acetate by Aldehyde Dehydrogenase (ALDH).

Acetaldehyde shortens telomere length, inhibits DNA repair mechanisms, and permanently damages DNA strands. Alcohol causes scavenger systems to malfunction and increases the production of ROS, which increases oxidative stress and contributes to genomic instability. A key relationship between drinking alcohol and the development of gastrointestinal cancer is the microbiome.

Microbiome of the mouth, oesophagus, and production of acetaldehyde

A risk factor for oral and oesophageal carcinogenesis is poor oral health. The upper digestive tract mucosa's exposure to acetaldehyde is significantly influenced by the local microbiota. A dysbiotic oral microbiome promotes the growth of opportunistic pathogens (like Candida yeasts) and can increase local acetaldehyde production by up to 100%. The expression of ethanol- and acetaldehyde-metabolizing enzymes is further altered by organ-specific gene polymorphisms and organ-specific expression patterns. Saliva and oral bacteria, which both play crucial roles in the pathogenesis of alcohol-related upper digestive tract malignancies, connect the oropharynx to the oesophagus and stomach. Millions of drinkers have been randomly exposed to noticeably more local acetaldehyde exposure through saliva and stomach juice due to a point mutation in the ALDH2 gene, increasing their risk of upper digestive tract malignancies significantly.

The oropharynx offers a special gene-epidemiologic and gene-biochemical human cancer paradigm for long-term local acetaldehyde exposure due to a single point mutation in the ALDH2 gene. The model firmly establishes the causative relationship between local acetaldehyde and gastric, oesophageal, and throat cancers, in addition to oropharyngeal cancer caused by alcohol. Regarding this gene mutation-based paradigm, it is significant to highlight that individuals with homozygotic ALDH2-deficiency (zero ALDH2 activity) typically are unable to consume alcohol at all due to uncomfortable side effects, including severe "Antabuslike" flushing. However, many heterozygotic ALDH2-deficient individuals (with some ALDH2 activity) use alcohol, some of whom even do so excessively or become alcoholicsIf so, they run a dose-dependently elevated risk of getting gastric, oesophageal, and oropharyngeal cancer. When they consume ethanol, their salivary and gastric juice levels of acetaldehyde are both increased by a factor of two and five, respectively. Oropharyngeal cancer has the highest cumulative ethanol-associated cancer risk per standard drink/day (12 g of

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ethanol/day). A person would, at best, never use tobacco, drink alcohol, or do both. However, even quitting smoking and consuming alcohol in moderation are linked to a significant reduction in local acetaldehyde exposure and the risk of developing alcohol-related cancer, particularly among established risk groups.