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Commentary

Concurrent Drinking and Smoking Synergistically Interact to Further Increase the Risk of Cancer

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DESCRIPTION

Two modifiable life-style factors, smoking and drinking are the leading causes of death leads to cancer. Excessive alcohol intake is associated with cancer of the upper digestive tract (larynx, esophagus, pharynx and oral cavity), liver and colon. Smoking rise the risk of esophageal cancers, mouth, and oropharyngeal cancer as well as cancer of the trachea, bronchus, lung, liver, stomach and urinary bladder. Concurrent drinking and smoking synergistically interact to further increase the risk of cancer.

Genetic variations in enzymes that metabolize alcohol and acetaldehyde and their relationships to risk for different types of cancer

Formation of reactive oxygen species, including superoxide, hydrogen peroxide and hydroxyl radicals and depletion of mitochondrial glutathione due to alcohol metabolism

DNA-acetaldehyde-adduct formation following alcohol metabolism and smoking, a critical event in carcinogenesis, and adducts involving DNA and hydroxyethyl radicals, as well as immunogenic adducts due to acetaldehyde

Tobacco smoking and cancer due to acetaldehyde and pro-carcinogens that are activated by CY P2E1 and CY P IA2. Alcohol's consequence on methionine and folate cycles that leads to hypomethylation of DNA which has been linked to an increase in cancer risk.

Life-style factors such as diet, alcohol consumption and cigarette smoking are important factors of disease including several types of cancer. Excessive alcohol consumption and smoking, two potentially modifiable risk factors, are the leading risk factors for death from cancer worldwide. A causal association has been reported between chronic alcohol consumption and cancer of the Upper Aerodigestive Tract (UADT, esophagus. pharynx, larynx, and oral cavity), liver, and colon; for pancreatic and lung cancer an association is suspected. Heavy drinking increases the risk for cancer. For example, the relative risk for esophageal cancer was 5.8 following daily consumption of 72 g about 6 drinks) of alcohol, and that for oral cancer, oropharyngeal cancer, and hypopharyngeal carcinoma was 13.5, 15.2, and 28.6, respectively. When more than 100 g of alcohol was consumed daily. In women, chronic alcohol is associated with increased risk for breast cancer. Tobacco smoking is associated with trachea, bronchus and lung cancers, as well as mouth and oropharyngeal, esophageal, stomach, liver, and bladder cancers. Simultaneous smoking and drinking further increases the risk for cancers of the UADT.

A number of comprehensive reviews on alcohol and cancer have been published and several mechanisms have been proposed for alcohol-induced cancer, including: formation of acetaldehyde; production of reactive oxygen species (ROSI and lipid peroxidation products changes in folate and methionine metabolism alcohol-induced increase in estrogen formation in breast cancer suppressed immune function; and alcohol's solvent action increase the bioavailability of carcinogens from tobacco and other sources.

The induction of microsomal cytochrome P450 enzymes by alcohol increases the metabolism of pro-carcinogens, such as nitrosamines, present in tobacco smoke, and may play an important role in the greater risk for cancer due to heavy alcohol consumption and smoking. This article focuses on ethanol metabolism and how genetic variations in the enzymes responsible for ethanol degradation increase cancer risk. It also addresses the roles of ROS and acetaldehyde in tumors due to alcohol and smoking, as well as folate and methionine metabolism, and their possible contribution to alcohol-induced cancer.

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