Abstract

Alcohol (ethanol) enters the human body mainly through ingestion of alcoholic beverages and its chronic consumption is considered a worldwide socio-economic problem. Besides others, alcohol consumption increases the risk of development of breast, liver, colorectal and upper aerodigestive tract cancer. In the liver, ethanol is metabolised into toxic acetaldehyde which is the main cause of DNA damage leading to cancer development. Acetaldehyde covalently interacts with nucleotides in the DNA forming DNA adducts such as N²-ethylidene-2′-deoxyguanosine or S- and R-α-methyl-γ-hydroxy-1,N²-propano-2′-deoxyguanosine. Acetaldehyde can also interact with proteins and disrupt their function. Ethanol metabolism by cytochrome P450 2E1 leads to production of reactive oxygen species, that subsequently damage cellular molecules such as lipides and DNA. Ethanol also initiates carcinogenesis through aberant DNA methylation or interference with retinoic acid metabolism. In cancer development, alcohol interacts with other environmental and genetic factors, which can increase the risk of developing cancer in predisposed individuals.