Abstract

Epidemiologic studies addressing the association of alcohol consumption with breast cancer consistently suggest a modest association and a dose-response relationship. The epidemiologic evidence does not point to a single mechanism to explain the association, and several mechanisms have been proposed. Alcohol consumption is shown to increase levels of endogenous estrogens, known risk factors for breast cancer. This hypothesis is further supported by data showing that the alcohol-breast cancer association is limited to women with estrogen-receptor positive tumors. Products of alcohol metabolism are known to be toxic and are hypothesized to cause DNA modifications that lead to cancer. Recent research has focused on genes that influence the rate of alcohol metabolism, with genes that raise blood concentrations of acetaldehyde hypothesized to heighten breast cancer risk. Mounting evidence suggests that antioxidant intake (e.g. folate) may reduce alcohol-associated breast cancer risk, because it neutralizes reactive oxygen species, a second-stage product of alcohol metabolism. Diets lacking sufficient antioxidant intake, as a result, may further elevate the risk of breast cancer among alcohol consumers. Given that alcohol consumption is increasing worldwide and especially among women in countries of rapid economic growth, a greater understanding of the mechanisms underlying the known alcohol-breast cancer association is warranted. Avoiding overconsumption of alcohol is recommended, especially for women with known risk factors for breast cancer.

Keywords

Breast cancer, alcohol consumption, risk factors.