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Alcohol dehydrogenase 3 and risk of esophageal and gastric adenocarcinomas.

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OBJECTIVES: Alcohol increases esophageal squamous carcinoma risk but has been less consistently associated with esophageal adenocarcinoma. Alcohol dehydrogenase catalyzes the oxidation of approximately 80% of ethanol to acetaldehyde, a carcinogen. The alcohol dehydrogenase gene has several polymorphisms which may lead to faster conversion of ethanol to acetaldehyde, which may increase cancer risk. METHODS: We undertook a study to examine whether a common polymorphism in the alcohol dehydrogenase 3 gene was associated with a higher risk of esophageal adenocarcinoma using data and biological samples collected for the Esophageal and Gastric Cancer Study (n = 114 esophageal and gastric cardia adenocarcinoma, n = 60 non-cardia gastric carcinoma, n = 23 cases of esophageal squamous cell carcinoma and 160 controls). RESULTS: Individuals homozygous for ADH (3) (1-1) had a higher risk of each tumor type compared to individuals who had ADH (3) (2-2) or ADH (3) (1-2) genotype (OR = 1.7, 95% CI = 1.0-2.9 for esophageal and gastric cardia adenocarcinomas; OR = 1.7, 95% CI = 0.7-4.3 for esophageal squamous cell carcinoma; and OR = 2.8, 95% CI = 1.5-5.1 for non-cardia gastric cancer). The elevation in risk from homozygosity of the ADH (3) (1) allele was seen in drinkers and nondrinkers, although the risk estimate was only significant for drinkers, particularly of liquor. CONCLUSION: These data suggest ADH3 genotype may be associated with risk of esophageal and gastric cardia adenocarcinomas.

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