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제 목	알콜탈수소효소 및 알데히드탈수소효소의 유전적 다형성이 대장암 발병위험에 미치는 영향 The effect of alcohol and aldehyde dehydrogenase polymorphisms on the risk of colorectal cancer
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Purpose: Alcohol and aldehyde dehydrogenase(ADH/ALDH) are major enzymes responsible for the metabolism of ethanol and acetaldehyde in the body. ALDH2*1/2*2 genotype, which encodes inactive ALDH2, and less active form of ADH2(ADH2*1/2*1) are known to enhance the risk for esophageal cancer, esp among heavy drinkers in East Asians. Inactive mutant allele of ALDH2*2 was found to be related with alcohol flushing response and to protect against alcoholism among Japanese. A multicenter case-control study was conducted to investigate the association of these polymorphisms and associated factors to colorectal cancer.

Methods: Cases were a consecutive series of patients with histologically confirmed, incident colorectal cancer who were admitted to two university hospitals and one cancer general hospital in Seoul, Korea between 1998 and 2004, and controls were selected at the same hospitals. A total of 810 cases and 846 controls were enrolled. Trained nurse interviewers collected information on smoking habit, alcohol intake, diet, and other lifestyle factors using a structured questionnaire. Subjects were genotyped for ADH/ALDH by polymerase chain reaction- CTPP methods.

Results: After considering potential covariates, cases were found to consume more alcohol (aOR=1.68, 95% CI 1.15-2.66), to be more likely current smokers (aOR=1.41, 95% CI 0.98-1.84), to eat more fat on meat (aOR=1.82, 95% CI 1.30-2.72), to eat dairy products less (aOR=0.65, 95% CI 0.38-0.92), and to participate vigorous physical activity less likely(aOR=0.77, 95% CI 0.57-0.98) than the controls. Those with variant GG genotype of ADH showed marginally increased risk for colorectal cancer(aOR=1.60, 95% CI 0.89-2.85), compared with those homozygous AA genotypes. The effect of GG genotype, however, was strongly pronounced among heavy drinkers consuming more than 30g ethanol per day (aOR=6.29, 95% CI 0.86-32.5), compared with non-drinkers (aOR=1.17, 95% CI 0.60-2.70). For ALDH2, The effect of alcohol was stronger among those with variant alleles (OR 3.21, 95% CI 1.04-5.84) than among those with wild type (OR 1.10, 95% CI 0.78-1.77).

Conclusion: There appears to be interactive effect between ADH/ALDH polymorphisms and alcohol consumption on the risk of colorectal cancer. Further analyses are underway.