번호 10-4

제목	Can Alcohol accelerate to develop breast cancer in women with GST null genotypes?		
저자 및 소속	Sue Kyung Park1, Keun-Young Yoo1, Seong-Jun Lee1, Lee CK1, Se-Hyun Ahn3, Dong-Young Noh2, Kuk-Jin Choe2, Daehee Kang1 *1Department of Preventive Medicine & 2General Surgery, Seoul National University College of Medicine, 3Department of General Surgery, College of Medicine, Ulsan University*		
분 야	보건관리( ), 역학(◎), 환경( )	발표자	일반회원 (◎), 전공의 ( )
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Background/Objectives *:* The null genotypes of glutathione S-transferase M1 (GSTM1) and GSTT1 might be associated with increased risk of breast cancer. Limited numbers of studies have been conducted to assess the increase/decrease the breast cancer risk in women with a genetically susceptible gene like GSTM1/T1 null type. The goals of this study were to evaluate whether breast cancer risk could be increase when women with genetically susceptible gene drinked alcohol.

Methods *:* A hospital based case-control study was conducted. Two hundreds and four histologically confirmed incident breast cancer cases with blood sample, and 332 controls with blood sample and no present or previous history of cancer were recruited from two teaching hospitals in Seoul during 1994 - 1998. One hundred and eighty nine cases and 233 controls were eligibly remained after excluding women with amenorrhea, benign breast mass (n=60), other breast diseases (e. g., mastitis, benign calcification, etc.), hormone-related diseases such as thyroid problems, and other systemic problems like chronic liver diseases and so on. By age category, each cases were finally matched to one controls and all subjects (189 cases and 189 controls) analyzed for GSTM1, GSTT1 genetic polymorphisms. GSTM1 and GSTT1 genetic polymorphism were determined for 181 breast cancer cases, and 188 controls by multiplex PCR. Forty samples were genotyped twice to evaluate the assay reliability. Odds ratios and 95% confidence intervals were estimated by unconditional logistic regression model, after adjustment for alleged risk factors of breast cancer.

Results *:* When data were stratified for alcohol consumption, the homozygous GSTM1 null genotype was a significant association with breast cancer risk(All women, OR=3.5, CI=1.35-8.98, premenopausals, OR=5.7, CI=1.85-17.61), but in postmenopausal women the relationship were not observed(OR=0.7, CI=0.37-1.37). The homozygous GSTT1 null genotype had not significant association with breast cancer in overall, premenopausal, and postmenopausal women. When data were stratified for alcohol consumption, the risk of breast cancer increased as the number of null GST genotypes increased (p for trend=0.03). For premenopausal women, when breast cancer risk of a women with GSTM1 positive & never-drinker is based on OR=1, risk of a women with GSTM1 null & never-drinker is evaluated as OR=1.4 (CI=0.7-3.0), with GSTM1 positive & ever-drinker as OR=0.9 (CI=0.3-2.3), and with GSTM1 null & ever-drinker as OR=5.5, (CI=1.7-18.0). For postmenopausal women, when breast cancer risk of a women with GSTM1 positive & never-drinker is based on OR=1, risk of a women with GSTM1 null & never-drinker is evaluated as OR=1.0 (CI=0.5-2.3), with GSTM1 positive & ever-drinker as OR=4.8 (CI=0.9-25.2), and with GSTM1 null & ever-drinker as OR=2.0 (CI=0.4-11.7).

Conclusions *:* These results suggest that for postmenopausals alcohol can independently affect breast cancer, but for premenopausals alcohol can synergistically affect breast cancer in only genetically susceptible individuals. Alcohol might be feasible risk factor for breast carcinogenesis.

Key words: GST genetic polymorphism, Alcohol, breast neoplasms