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제 목	GPX1 및 hOGG1 유전자다형성에 따른 유전자의 산화적 손상 및 폐암 발생 위험도 평가 Effects of oxidative DNA damage and genetic polymorphism of the glutathione peroxidase (GPX)1 and 8-oxoguanine glycosylase 1 (hOGG1) on lung cancer
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Objectives: Oxidative DNA damage is a known risk factor of lung cancer. The glutathione peroxidase (GPX), antioxidant enxyme that reduce hydrogen peroxide and lipid peroxides, plays a significant role in the protection of cells from oxidative stress induced by reactive oxygen species. The aim of this case-control study was to investigate effects of oxidative stress, genetic polymorphisms of GPX1 genes and interaction between them in the carcinogenesis of lung cancer.

Methods: Two hundreds patients with lung cancer and 200 age- and sex-matched controls were enrolled in this study. Every subject was asked to complete a questionnaire concerning their smoking habits and environmental exposure to PAHs. Genotype of GPX1 and 8-oxoguanine glycosylase 1 (hOGG1) genes were examined and, concentrations of urinary 1-hydroxypyrene (1-OHP), 2-naphthol, and 8-hydroxydeoxyguanosine (8-OH-dG) were measured.

Results: Cigarette smoking was a significant risk factor for lung cancer. Levels of urinary 8-OH-dG were higher in patients ($p<0.001$), whereas urinary 1-OHP and 2-naphthol levels were higher in controls. GPX1 codon 198 polymorphism was associated with an increased risk of lung cancer. Individuals carrying Pro/Leu or Leu/Leu genotype of GPX1 were at higher risk of lung cancer (Adjusted OR=2.29). In addition these individuals shown to have high level of urinary 8-OH-dG concentrations compared to individuals with the GPX1 Pro/Pro genotype.

Conclusion: These results lead to a conclusion that individuals with the GPX1 Pro/Leu or Leu/Leu genotype would be more susceptible to lung cancer induced by oxidative stress than those with Pro/Pro genotype.

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