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Association between environmental tobacco smoke exposure and lung cancer susceptibility: Modification by antioxidant enzymes genetic polymorphisms

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Background: Environmental Tobacco Smoke (ETS) is the primary etiological factor of lung cancer. However, only 10-15% of smokers develop lung cancer suggesting genetic role in modifying individual susceptibility to lung cancer. Antioxidant enzyme functional genetic polymorphisms should be considered.

Aim: The present study aimed to evaluate the role of antioxidant enzyme activity and genetic polymorphisms in modifying the susceptibility to lung cancer among individuals exposed to ETS.

Subjects & Methods: A total of 150 male subjects were divided into three groups: 50 lung cancer patients, 50 chronic smokers and 50 passive smokers. Genotyping of mEH exon 3 (Tyr113Hist) and exon 4 (Hist139Arg) polymorphisms was done by PCR–RFLP technique. MnSOD (Val16Ala) polymorphism was detected by Real time-TaqMan assay. Erythrocyte MnSOD activity was measured spectrophotometrically.

Results: ETS exposed individuals (both active and passive smokers) who carried His allele of mEH exon3 have 2.9-folds increased risk of lung cancer (OR 2.9 $P<0.001$). Also ETS exposed carriers of Arg allele of mEH exon 4 have 2.1-folds higher risk to lung cancer (OR 2.1 $P=0.024$). However no association between MnSOD Val16Ala polymorphism and lung cancer was detected among ETS (OR 1.6 $P=0.147$), although lung cancer group had significantly lower MnSOD activity than chronic or passive smokers groups ($P=0.03$).

Conclusion: Exons 3 and 4 polymorphisms of the mEH gene may contribute to lung cancer susceptibility through disturbed antioxidant balance. However, this was not the case with MnSOD Val16Ala SNP. Antioxidant enzymes may modulate the influence of ETS exposure on lung cancer risk.

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