ASBESTOSIS AND GENE-ENVIRONMENT INTERACTIONS

1A Franko, 2Y Dolzan, 3M Dodic-Filip. 1Clinical Institute of Occupational Medicine, University Medical Centre, Ljubljana, Slovenia; 2Pharmacogenetics Laboratory, Institute of Biochemistry, Faculty of Medicine, University of Ljubljana, Slovenia

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Introduction

Although the causal relationship between asbestosis and asbestos exposure has been well proved, little is known about the genetic factors that may influence the development of this disease. This study investigated the influence of gene-gene and gene-environment interactions on the risk of developing asbestosis.

Methods

The nested case-control study included 262 cases with asbestosis and 265 controls with no asbestos-related disease studied for MnSOD, ECOSOD, CAT, GSTT1, GSTM1, GSTP1, and iNOS polymorphisms. Data on cumulative asbestos exposure and smoking were available for all subjects. PCR-based methods were used for genotyping. Logistic regression analysis was used to assess asbestosis risk.

Result

The OR of asbestosis was 3.21 (95% CI: 2.43 to 4.23) for cumulative asbestos exposure; 0.98 (95% CI: 0.69 to 1.39) for smoking; 1.50 (95% CI: 1.01 to 2.24) for MnSOD 9Ala/Ala versus Ala/Val and Val/Val; 1.63 (95% CI: 0.62 to 4.27) for ECOSOD 213Arg/Gly versus Arg/Arg; 1.36 (95% CI: 0.70 to 2.62) for CAT –262TT versus CT and CC; 1.20 (95% CI: 0.85 to 1.69) for iNOS LL versus SL and SS; 1.01 (95% CI: 0.71 to 1.43) for GSTM1-null; 0.61 (95% CI: 0.40 to 0.94) for GSTT1-null; 1.52 (95% CI: 1.08 to 2.15) for GSTP1 105Ile/Ile versus 105Ile/Val and 105Val/Val; and 0.97 (95% CI: 0.64 to 1.48) for GSTP1 114Ala/Ala versus 114Ala/Val and 114Val/Val. The associations between MnSOD Ala–9Val polymorphism and asbestosis, and between iNOS genotypes and asbestosis (p=0.038) were modified by CAT–262C>T polymorphism (p=0.031). A strong interaction was found between GSTM1-null polymorphism and smoking (p=0.007), iNOS (CCTTT)\textsubscript{n} polymorphism and smoking (p=0.054) as well as between iNOS (CCTTT)\textsubscript{n} polymorphism and cumulative asbestos exposure (p=0.037).

Discussion

The findings suggest that in addition to asbestos exposure the interactions between different genotypes, genotypes and smoking, and between genotypes and asbestos exposure have an important influence on developing asbestosis and should be considered seriously in future research on occupational/environmental asbestos-related diseases.