Genetic-biochemical Criteria for Individual Sensitivity in Development of Occupational Bronchopulmonary Diseases

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SUMMARY

Human individual sensitivity to health-hazardous occupational factors and probability of developing chronic lung diseases depend on genetic variation of serum and erythrocytic proteins. The present work was aimed at studying the phenotypes of serum and erythrocytic proteins in patients with occupational respiratory diseases. We studied 7 highly polymorphic genetic systems the varieties of which may be connected with development of bronchopulmonary pathology (BPP) and the immune status of the body: proteinase inhibitor (Pi), third component of the complement (C3), transferrin (Tf), group-specific component of blood serum (Gc), haptoglobin (Hp), erythrocytic glyoxalase (Glo) and phosphoglucomutase (PGM) in patients with chronic bronchitis, silicosis, occupational bronchial asthma and in the control group consisting of Moscow population not exposed to occupational hazards and apparently healthy workers of an engineering plant.

Considerable differences were revealed in genetic structure of the patients with bronchopulmonary pathology as compared with the apparently healthy people along a series of Integrated system: proteinase inhibitor (Pi), C3, Tf, Gc, PGM. Comparison of the study groups by significant differences in the aggregate of the genetic information obtained suggests that 5 (HP, C3, Tf, PI, PGM1) of the 7 studied systems showed the hereditary features of silicosis. The gene carriers Hp*2, C3*F, PGM1*2-, TF*D, GC*R due to peculiar biochemical processes appear to have less adaptive potentialities and a greater likelihood of the disease on exposure to industrial factors. The examined patients with chronic bronchitis showed an increase in the variant of GC*2 and of a rare variants of proteins GC*R and Pi*S, the patients with occupational bronchial asthma showed an increase in the variant of Hp*2 and of a rare variant Pi*S.

Such studies could be useful for assessment and forecast of individual risk of occupational diseases.

Key words: silicosis, bronchitis, bronchial asthma, polymorphism

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