Smoking as a risk factor which activates IL-2 gene polymorphism in patients with MDR-TB

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Background. Determine IL-2 gene polymorphism (PM) in smokers with MDR-TB in lungs on the background of the respective cytokine production of blood. Methods. The study included 170 people in Kharkiv region of Ukraine including 60 patients with MDR-TB and smoking (1st group), 36 without MDR-TB and smoking (2nd group), 14 with MDR-TB and non-smoking (3rd group), 30 without MDR-TB and non-smoking (4th group), and 30 healthy donors (5th group). Studied promoter region T-330G of IL-2 gene.

Results. In the 1st group the levels of IL-2 were (41.24±1.21), 2nd group (39.81±1.17), 3rd group (31.19±1.90), 4th group (31.87±0.84) and 5th group (21.60±0.80) pg/L. Differences between groups were significant (p<0.001). Normal homozygote genotype IL-2 predominated in 5th group was 60% compared to patients with TB: 1st group - 5%, 2nd group - 13.89%, 3rd group - 14.29% and 4th group - 16.67%. Heterozygous genotype IL-2 was observed in 76.67% in 1st, 45.71% in 2nd, 35.71% in the 3rd, 13.33% in 4th and 16.66% in 5th group. Mutation homozygous genotype IL-2 was 18.33% in the 1st group, 2nd group - 41.67%, 3rd group - 50%, 4th group - 70%, and 5th group - 3.34%. Differences between the 1st and 3rd, 2nd and 4th, TB patients and 5th group were significant (p<0.05).

Conclusion. Smoking contributes to significant activation PM T-330G gene IL-2 heterozygous type that may lead to changes in the immune system making susceptible to MDR-TB. Compared to healthy controls patients with TB had significantly increased levels of serum IL-2. This coincided with greater frequency of heterozygous PM T-330G of IL-2 gene. In addition, these studies revealed a significant influence of the PM T-330G gene IL-2 (with PM IL-4 and IL-10 genes, which we also studied) on the changes in the population of Th-lymphocytes, clinical symptoms, relapse of TB, formation destructions in the lung, which may treatment outcomes in patients with MDR-TB.

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