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ASSOCIATION OF TP53 PRO72ARG GENE POLYMORPHISM WITH THE RISK OF BENIGN PROSTATIC HYPERPLASIA

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Abstract

Background. *Benign prostatic hyperplasia (BPH) is one of the most prevalent urological disorders among aging men. Although hormonal and metabolic factors contribute significantly to disease development, increasing attention has been focused on the role of genetic polymorphisms involved in cellular proliferation, apoptosis, and DNA repair. The tumor suppressor gene* ***TP53*** *plays a central role in maintaining genomic stability by regulating cell-cycle arrest, apoptosis, and DNA damage repair. Functional polymorphisms of this gene may alter its biological activity and increase susceptibility to prostate diseases.*

Keywords: *TP53, Pro72Arg polymorphism, benign prostatic hyperplasia, molecular genetics, genetic susceptibility, biomarkers, prostate disease.*

Objective. To evaluate the association between the TP53 Pro72Arg polymorphism and the risk of benign prostatic hyperplasia.

Materials and Methods. A case-control study included 74 patients diagnosed with benign prostatic hyperplasia and 105 healthy male controls. Genotyping of the TP53 Pro72Arg polymorphism was performed using molecular genetic techniques. Allele and genotype frequencies were compared between study groups, and Hardy–Weinberg equilibrium analysis was performed. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to estimate genetic risk.

Results. In patients with BPH, the Pro allele represented 85.8% of all alleles, whereas the Arg allele accounted for 14.2%. In the control group, the Pro allele frequency reached 93.3%, while the Arg allele frequency was only 6.7%.

Genotype analysis demonstrated that the Pro/Pro genotype was observed in 74.5% of patients, the Pro/Arg genotype in 22.6%, and the Arg/Arg genotype in 2.8%. In contrast, the control group showed Pro/Pro and Pro/Arg frequencies of 86.7% and 13.3%, respectively, while the Arg/Arg genotype was not detected.

Comparative analysis revealed a significantly higher frequency of the Arg allele among BPH patients (OR = 2.3; 95% CI: 1.20–4.43; $p = 0.03$), indicating that carriers of this allele have an increased genetic susceptibility to BPH. Conversely, the Pro allele demonstrated a protective effect against disease development. Although some genotype comparisons did not reach statistical significance, the heterozygous Pro/Arg genotype tended to occur more frequently among affected individuals, suggesting its potential contribution to disease initiation.

These findings support the hypothesis that TP53 genetic variation influences the biological mechanisms underlying prostatic hyperplasia through impaired regulation of apoptosis and cellular proliferation.

Conclusion. The TP53 Pro72Arg polymorphism appears to be associated with susceptibility to benign prostatic hyperplasia. The Arg allele and Pro/Arg genotype may serve as promising molecular biomarkers for identifying individuals at increased genetic risk. Incorporating TP53 genotyping into clinical practice may improve early diagnosis, individualized risk prediction, and preventive strategies for patients predisposed to prostate diseases.