

Plasmatic Variations of Metalloproteinase 9 (MMP-9) Due to Functional Polymorphism in Prostate Cancer

Introduction and Objectives: Matrix metalloproteinase 9 (MMP-9) is an endopeptidase related to the development of prostate cancer. The -1562C/T polymorphism of MMP-9 increases the risk of suffering from prostate cancer. The -1562C/T polymorphism is found in the promoter region of MMP-9 and its presence implies the loss of the binding site of repressor transcriptional protein. The studies which analyze the relationship of the -1562C/T polymorphism with cancer are based on the fact that the presence of -1562C/T polymorphism increases the transcription of MMP-9 and thus the plasmatic levels of MMP-9. However, there is no data about the association between the plasmatic levels of MMP-9 and the presence of -1562C/T polymorphism in prostate cancer patients. The aim of this study is to analyze the plasmatic levels of MMP-9 concerning the genotypes of -1562C/T polymorphism in patients who may have prostate cancer.

Materials and Methods: The prospective cohort was made up of 235 patients. All the patients were subjected to a prostatic biopsy and a blood extraction. The identification of the genotype of the -1562C/T polymorphism was carried out by means of RFLP (restriction fragment length polymorphism). The plasma determination of MMP-9 was carried out by means of immunoassay.

Results: There were found to be 90 prostatic tumors. Genotypical frequency was 80% wild homozygous, 18% heterozygous, 2% polymorphic homozygous. The average plasmatic concentration of MMP-9 was 498 ng/ml. No statistically significant differences were found when comparing the plasmatic concentration of MMP-9 and the genotype of -1562C/T polymorphism ($p=0.611$). We repeated the analysis using subgroups of prostatic biopsy results and no statistically significant differences appeared either (tumorous subgroup $p=0.692$; Non-tumorous subgroup $p=0.669$). Likewise, no statistically significant differences were found when comparing plasmatic concentration of MMP-9 with prostatic biopsy results, based on the genotype of -1562C/T polymorphism (wild homozygous $p=0.499$; heterozygous $p=0.996$; polymorphic homozygous $p=0.594$).

Conclusions: The -1562C/T polymorphism does not alter the plasmatic levels of MMP-9 in prostate cancer patients, nor in patients showing no cancer in the prostatic biopsy. Therefore, the action mechanism of the -1562C/T polymorphism in the development of prostate cancer does not increase the levels of MMP-9 in plasma.