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**Introduction & Objectives:** Gene expression levels of STK11 and PRKACA were investigated to determine their possible correlation with benign and malignant prostate diseases and the related clinical parameters. Also, their diagnostic capability to differentiate benign versus malignant prostatic disease was sought out.

**Materials & Methods:** Two groups of sixty patients each operated for benign prostatic hyperplasia (BPH) or clinically localized prostate cancer between January 2016 and June 2017, were included in the study. The G6PD gene was used as the housekeeping gene. Gene expression analyses of all STK11, PRKACA and G6PD genes were performed using fresh histopathologically diagnosed hyperplastic or cancerous prostate tissues together with normal counterparts, from all patients. The correlations of STK11 and PRKACA expressions with the clinicopathologic parameters of the patients were studied to clarify their role in the pathophysiology and potential role as a diagnostic marker.

**Results:** Relevant gene expressions could be determined in 50/60 of patients with BPH and 57/60 of patients with localized prostate cancer. STK11 gene expression was increased and PRKACA gene expression decreased in approximately 60% of both BPH and localized prostate cancer tissues. However, no significant correlation was found between expression of the two genes. On the other hand, regardless of pathology, a correlation between prostate volume and expression was noted for both genes. In larger prostate tissues PRKACA gene expression was significantly increased ( $p=0,01$ ) while STK11 gene expression significantly decreased ( $p=0,046$ ). When qPCR parameters were evaluated a significant difference between the cycle threshold (Ct) values of STK11 was observed for BPH and prostate cancer. When all clinical parameters, laboratory values and qPCR parameters were evaluated using ROC analyses to predict prostate cancer (Figure), the ratio of PSA Density/cycle threshold for STK11 {AUC=0,895  $p=0,001$  (95% CI 0,836-0,953)} was found to predict prostate cancer more effectively when compared to PSA density or PSA alone.

**Conclusions:** Similar but inverse rates of changes in the expression of STK11 and PRKACA genes were observed for both BPH and localized prostate cancer. Expression of STK11 and PRKACA genes were correlated with the volume of the prostate. In the ROC analyses, the ratio defined by the PSA Density/cycle threshold for the STK11 gene was found as an effective parameter to predict prostate cancer.