AB077. The expression and clinical significance of α-actinin-1 in prostate cancer and benign prostatic hyperplasia

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Background: To investigate the expression and clinical significance of α-actinin-1 protein in prostate cancer and prostatic hyperplasia.
Methods: Collection all the clinical data of the patients of prostate cancer and prostatic hyperplasia treated in our school affiliated hospital between January 2007 and October 2014, according to certain criteria into a group filter. Immunohistochemistry method was used to detect the expression of α-actinin-1 in 30 cases of prostate cancer (PCa) tissues and 30 cases of benign prostatic hyperplasia (BPH) tissues. Western blot method was used to detect the relative expression of α-actinin-1 in 18 cases of PCa tissues and 20 cases of BPH tissues among the two groups.
Results: The result of Immunohistochemistry shows that the positive expression rate of α-actinin-1 in PCa and BPH was 76.7% and 20% respectively. The difference was statistically significant (P<0.01). And the result of Western Plot shows that the expression of α-actinin-1 in PCa tissues was significantly higher than that in BPH tissues. The difference was statistically significant (P<0.05). The expression of α-actinin-1 in PCa has statistically significant with Gleason scores and T staging (P<0.05) instead of age, serum PSA, whether there is bone metastasis, whether there is lymph node metastasis (P>0.05).
Conclusions: The expression of α-actinin-1 in PCa tissues was significantly higher than that in BPH tissues, and there may exist certain relationship with Gleason scores and T staging.
Keywords: Prostate cancer (PCa); benign prostatic hyperplasia (BPH); α-actinin-1

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AB078. Knockdown of SPC25 inhibits cell proliferation and cycle progression in prostate cancer

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Abstract: Prostate cancer (PCa) was one of the most commonly diagnosed malignant cancers in males in China. Cell-cycle aberration was a hallmark of cancer. SPC25, a component of Ndc80 complex, played an important role in regulating mitotic chromosome segregation. However, the functional roles of SPC25 in prostate cancer were still poorly understood. Our study showed for the first time that SPC25 was significantly upregulated in prostate cancer. To explore the molecular function roles of SPC25, we performed loss of function assay and found SPC25 knockdown inhibited cell proliferation and induced the decrease in S phase and the increase in G2/M phase. Furthermore, SPC25 knockdown promoted apoptosis of prostate cancer cells. Of note, bioinformatics analysis also revealed multiple functional roles of SPC25 in regulating cell proliferation, apoptosis, invasion, role of tissue factor in cancer, TGF-β signaling, and sumoylation pathway in PCa. Here, we also evaluated possible prognostic value of SPC25 using TCGA RNA-seq data and we found showed SPC25 was upregulated in high pathology stage PCa. Kaplan-Meier analysis showed that Lower SPC25 expression level was associated with better survival of PCa patients. All these results suggest that SPC25 play an oncogenic role in PCa and could act as a novel diagnostic and therapeutic target for prostate cancer.

Keywords: Prostate cancer (PCa); SPC25; prognosis; proliferation; cell cycle

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AB079. Upregulation of DEPDC1B correlates with tumor progression and predicts a poor prognosis in prostate cancer

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Abstract: One of the challenges in prostate cancer (PCa) treatment is lacking biomarkers that could accurately predict PCa progression. The most widely used biomarkers for PCa was prostate-specific antigen (PSA) level. However, PSA testing had low specificity for prostate cancer due to some other kinds of disease, such as PBH, could also induce PSA levels. Of note, PSA testing could not discriminate different stages of PCa. Although recently studies had identified a few genes including UHRF1, PCA3, PCAT-1 and PCAT-14showed PCa-associated dysregulation, there was still an urgent need to identify novel prognostic biomarkers for PCa. The DEPDC1B gene is located on chromosome 5. Previous studies indicated DEPDC1Bpalyed an important role in regulating cell cycle and migration. For example, Marchesi *et al*. found DEPDC1B was a cell-cycle-regulated gene by regulating the interplay between cell-cycle progression and de-adhesion events at the mitotic entry. In non-small cell lung cancer, ectopic expression of DEPDC1B enhanced migration and invasion of cancer cells via activating Wnt/β-catenin signaling. However, the clinical relevance and functional roles of DEPDC1B in PCa remain unclear. In this study, we found that the expression levels of DEPDC1B in PCa tissues were significantly higher than that in non-tumor tissues. Furthermore, our results showed DEPDC1B was upregulated in high pathology stage PCa. Kaplan-Meier analysis showed that Lower DEPDC1B expression level was associated with better survival of PCa patients. GO and KEGG pathway analysis of DEPDC1B co-expressed genes showed DEPDC1B played an important role in regulating PCa proliferation and cell cycle progression. We believed that this study will provide a potential new therapeutic and prognostic target for prostate cancer.

Keywords: Prostate cancer (PCa); DEPDC1B; prognosis; tumor progression; cell

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AB080. Annexin A5 regulates Leydig cell testosterone production via ERK1/2 pathway

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Abstract: This study was to investigate the effect of annexin A5 on testosterone secretion from primary rat Leydig cells and the underlying mechanisms. Isolated rat Leydig cells were treated with annexin A5. Testosterone production was detected by chemiluminescence assay. The protein and mRNA of steroidogenic acute regulatory (StAR), P450scc, 3β-hydroxysteroid dehydrogenase (3β-HSD), 17β-hydroxysteroid dehydrogenase (17β-HSD) and

