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

