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

17α-hydroxylase were examined by western blotting and RT-PCR, respectively. Annexin A5 significantly stimulated testosterone secretion from rat Leydig cells in dose- and time-dependent manners and increased mRNA and protein expression of StAR, P450scc, 3β-HSD and 17β-HSD but not 17α-hydroxylase. Annexin A5 knockdown by siRNA significantly decreased the level of testosterone and protein expression of P450scc, 3β-HSD and 17β-HSD. The significant activation of ERK1/2 signaling was observed at 5, 10, and 30 min after annexin A5 treatment. After the pretreatment of Leydig cells with ERK inhibitor PD98059 (50 μmol/L) for 20 min, the effects of annexin A5 on promoting testosterone secretion and increasing the expression of P450scc, 3β-HSD and 17β- HSD were completely abrogated (P<0.05). Thus, ERK1/2 signaling is involved in the roles of annexin A5 in mediating testosterone production and the expression of P450scc, 3β-HSD and 17β-HSD in Leydig cells.

Keywords: Annexin A5; testosterone; steroidogenic acute regulatory (StAR); P450scc; 3β-hydroxysteroid dehydrogenase (3β-HSD); 17β-hydroxysteroid dehydrogenase (17β-HSD)

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AB081. Research on epididymis inflammatory mass in 1,021 patients with varicocele

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Background: To study the morbidity situation of inflammatory mass in epididymis head of varicocele (VC) patients and explore the mechanism of VC causing male sterility.

Methods: In total, 1,021 cases of VC with male infertility including 989 cases of VC on the left side, 554 cases of VC on both sides and 8 cases of VC on the right side, divided in mild-to-moderate group and severe group respectively. And107 cases of male infertility without VC as control group. Compared the incidence and location of the epididymis inflammatory mass, the temperature of the scrotum, sperm viability (a + b)%, neutral a-glucosidase levels and sex hormone levels between these groups.

Results: The incidence of inflammatory mass on the left side of the epididymis head were 21.8% [167] in mild-to-moderate group of VC on the left side, 58.1% [54] in severe group of VC on the left side, 14.4% [20] in mild-to-moderate group of VC on both sides, 26.7% [4] in severe group of VC on both sides and 12.1% [13] in control group. The incidence of inflammatory mass on both sides of the epididymis head were 48.4% [371] in mild-to-moderate group of VC on the left side, 7.5% [7] in severe group of VC on the left side, 28.8% [40] in mild-to-moderate group of VC on both sides, 25% [3] in severe group of VC on both sides, 25% [2] in group of VC on the right side and 23.4% [25] in control group. The incidence of inflammatory mass on the epididymis head in cases of VC with male infertility was higher than that in cases of male infertility without VC (P<0.05). The incidence of inflammatory mass on the cauda epididymis was 3.0% [23] in mild-to-moderate group of VC on the left side, 0.6% [8] in severe group of VC on the left side, 2.0% [3] in mild-to-moderate group of VC on both sides, 13.3% [2] in severe group of VC on both sides and 7.5% [8] in control group. VC patients with left and right scrotal temperatures higher than the control group, the difference was statistically significant (P<0.05). Sperm viability in VC patients with epididymis inflammatory mass, VC patients without epididymis inflammatory mass, the control group with epididymis inflammatory mass and the control group without epididymis inflammatory mass were 17.42±10.65, 34.71±12.31, 20.45±8.29 and 35.63±8.75, respectively. neutral a-glucosidase levels in these four groups were 19.13±5.62, 34.82±7.51, 26.47±5.62, 46.38±9.27, respectively. Sperm viability and neutral a-glucosidase levels in VC patients with epididymis inflammatory mass were significantly lower than other groups (P<0.05).

Conclusions: The morbidity rate of epididymis inflammatory mass in VC patients was significantly higher. Epididymis inflammatory mass was direct cause that VC lower sperm motility and glycosidic levels.

